DERM Technical Report:

Development of Cleanup Target Levels (CTLs)

for

Chapter 24, Code of Miami-Dade County, Florida

Prepared for the

Department of Environmental Resources Management (DERM), Miami-Dade County, Florida

by

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I. Introduction

This document describes the procedures used to develop soil, groundwater and surface water Cleanup Target Levels (CTLs), provides the equations used for calculating these values, and identifies the sources of input values for these equations. In addition, this document presents information regarding the derivation of site-specific soil CTLs, including methodology for selection of the appropriate input values for their calculation.

Groundwater CTLs are developed for the protection of human health based on the numerical standards in Section 24-12(2)(H) of the Code of Miami-Dade County and Chapter 62-550, Florida Administrative Code (F.A.C.), *Drinking Water Standards, Monitoring, and Reporting*, and the methodology provided in Chapter 62-777, F.A.C.. Freshwater and marine surface water CTLs are developed for the protection of human health and protection of aquatic species based on the numerical standards in Section 24-11(4) of the Code of Miami-Dade County and the methodologies employed in Chapter 62-302, F.A.C. and Chapter 62-777, F.A.C.

The approach in calculating soil CTLs described herein is consistent with that employed by the Florida Department of Environmental Protection (FDEP) for setting Soil Cleanup Target Levels under Chapter 62-777, F.A.C. (Saranko et al., 1999). The methodologies described here and in Chapter 62-777, F.A.C. are largely based on earlier efforts made by USEPA (1996a, 1996b, 1998). The rationale for selecting specific methods for use in Miami-Dade County from these and other sources is discussed in this report. While an attempt has been made to provide a comprehensive description of methods for calculating Miami-Dade County soil CTLs, in some instances the reader is referred to the source document for a more exhaustive explanation.

Although soil CTLs for various exposure scenarios can be calculated using the methodology presented here, this report focuses on only two scenarios: exposure from residential and from commercial/industrial land use. Soil CTLs are based on default assumptions and are intended to be

broadly applicable. Site-specific characteristics can be used to develop site-specific CTLs. Methods for calculating these site-specific CTLs are discussed.

II. Development of Groundwater CTLs

A. Introduction

Groundwater CTLs are equivalent to the numerical standards set forth in Section 24-12(2)(H) of the Code of Miami-Dade County. Where such standards do not exist, the groundwater CTLs are equivalent to the numerical standards set forth in Chapter 62-550, F.A.C., and presented in Table 1 through Table 4 of that document. For chemicals not listed in Section 24-12(2)(H) or Chapter 62-550, F.A.C., groundwater CTLs are based on the following factors, as applicable: 1) human health risk calculations using a lifetime excess cancer risk level of one in one million (1×10^{-6}) and a hazard quotient of one (1) or less [Note: these are designated in Table 1 by the notation 'health-based criteria' below the standard]; and 2) aesthetic considerations [Note: these are designated in Table 1 by the notation 'organoleptic' below the standard]. Aesthetic considerations include altered taste, odor, or color of the water. While these factors do not pertain to health directly, they nonetheless degrade the potability of the water, and therefore its suitability as a drinking water source. Therefore, the groundwater CTLs presented herein are identical to those found in Chapter 62-777, F.A.C. except for those cases where numerical standards are specified in Section 24-12(2)(H) of the Code of Miami-Dade County (arsenic, cyanide and phenol) or where the value in Chapter 62-777, F.A.C. is based on a practical quantitation limit (PQL). Unlike Cleanup Target Levels for some chemicals listed in Chapter 62-777, F.A.C., all DERM groundwater CTLs are either health-based or based on aesthetic considerations, irrespective of their PQLs. Groundwater CTLs are shown in Table 1. For each value, a footnote is included indicating the source or basis for derivation of that number.

B. Human Risk Equation

The equation used to calculate risk-based groundwater CTLs for carcinogens is shown in Figure 1. The equation for calculating groundwater CTLs based on non-cancer health effects is shown in Figure 2. Both equations are identical to those used by FDEP in deriving risk-based groundwater minimum criteria. The following sections discuss input values used in the equations to calculate groundwater CTLs for Chapter 24. Examples of calculations are shown in Figures 1 and 2.

1. Risk versus Hazard

All non-cancer health effects are assumed to have a dose threshold. That is, it is assumed that below some dose, the effect does not occur. A chemical can often produce many different types of adverse health effects, each with its own threshold. If the threshold for the most sensitive health effect can be identified — the effect that occurs at the lowest dose — limiting exposure to produce doses below that threshold should protect against all of the effects of the chemical. This concept is the basis for the USEPA reference dose. The USEPA examines toxicity data for a chemical, identifies the most sensitive effect, and then determines a dose sufficiently low enough to prevent that effect from occurring in the most sensitive individuals. Because environmental exposures can be long term, the dose is actually a dosing rate (amount of chemical per day), and it is intended to protect against toxicity for exposures that range up to a lifetime. Reference doses are specific to the route of exposure (ingestion, dermal contact, or inhalation). Since the greatest potential exposure to contaminants in groundwater is from use of the groundwater as a potable drinking water source, the most relevant reference dose for groundwater CTL development is the oral reference dose (RfDo) based on ingestion. Although ingestion of contaminated groundwater is the most likely source of exposure, its use for showering and cooking may also contribute to the overall exposure of receptors in these scenarios.

For hazard calculations, the projected exposure dose divided by the applicable reference dose is termed the *hazard quotient*. Groundwater CTLs are calculated based on a hazard quotient of 1. This means that the chemical dose implicit in the standard is equivalent to the maximum safe oral dose developed for that chemical by the USEPA for lifetime exposure.

Regulatory agencies currently view risks from carcinogens differently from non-cancer health effects. Chemical carcinogenicity is assumed not to have a threshold, and even very small doses are assumed to pose some (albeit small) risk of cancer. In this view, safety must be defined as some risk (i.e., probability) of cancer so small as to be considered insignificant. For Chapter 24, an excess cancer risk of 1×10^{-6} (one in one million) is used for calculating groundwater CTLs for carcinogens. FDEP also uses 1×10^{-6} as the target excess cancer risk in deriving minimum criteria for carcinogens in groundwater. Chemical carcinogens vary in their potency in producing cancer, and thus groundwater standards corresponding to a 1×10^{-6} excess cancer risk will often be different for different chemicals. The USEPA has developed measurements of cancer potency of carcinogens, which are termed *cancer slope factors* (CSFs).

It is important to point out that the toxicity values developed by the USEPA — the reference doses and cancer slope factors — are developed conservatively. That is, in view of uncertainties in the risk assessment process, they typically have a "safety buffer" built in. As a result, it is more correct to state, for example, that a groundwater CTL represents a risk "that is likely no more than one in a million" rather than to state that they are "equal to one in a million."

There are some chemicals designated as Class C carcinogens (i.e., possible human carcinogens) for which no cancer slope factor is available. Without a cancer slope factor, a groundwater CTL based on cancer risk could not be calculated. Consistent with the approach used by FDEP, groundwater CTLs for these chemicals are developed by reducing the standards calculated for non-cancer health effects by an additional factor of 10. The equation used to calculate groundwater CTLs for Class C carcinogens without defined slope factors is shown below.

$$\text{Groundwater CTL} = \frac{\frac{RfD_o}{10} \bullet 0.2\ \text{RSC} \bullet 70\ \text{kg} \bullet 1000\ \mu\text{g/mg}}{2\ \text{L/day}}$$

where:

RfD_o = Oral Reference Dose (mg/kg day)

RSC = Relative Source Contribution (20% default)

The Class C carcinogens that have groundwater CTLs based on non-cancer health effects along with the applicable groundwater CTLs are shown in the Table below.

Groundwater CTLs for C Carcinogens Based on Non-Cancer Health Effects

Contaminant	CAS#	Oral RfD (mg/kg-d)	Groundwater CTL (µg/L)
acrolein	107-02-8	2.0E-2	14
allyl chloride	107-05-1	2.857E-4	3.5
butyl benzyl phthalate, n-	85-68-7	2.0E-1	140
dichloroethane, 1,1-	75-34-3	1.786E-1	1300
linuron	330-55-2	2.0E-3	1.4
mercuric chloride	7487-94-7	3.0E-4	0.2
mercury, methyl	22967-92-6	1.0E-4	0.07
methidathion	950-37-8	1.0E-3	0.7
methylphenol, 2-	95-48-7	5.0E-2	35
methylphenol, 3-	108-39-4	5.0E-2	35
methylphenol, 4-	106-44-5	5.0E-3	3.5
metolachlor	51218-45-2	1.5E-1	110
oryzalin	19044-88-3	5.0E-2	35
parathion	56-38-2	6.0E-3	4.2
trichloracetic acid	76-03-9	1.3E-2	9.1

2. Exposure Parameters

Groundwater CTLs are based on consumption of 2 L of water per day and a body weight of 70 kg. These are standard drinking water exposure assumptions recommended by the USEPA. Exposure is assumed to occur over a lifetime. For non-carcinogens, a Relative Source Contribution (RSC) factor is included. This represents the fraction of the total allowable intake that can come from groundwater. Consistent with USEPA methods, a default RSC of 0.2 (20%) is used.

3. Toxicity Values

Calculation of a risk-based groundwater CTL requires a chemical-specific toxicity value, either an RfDo or an oral cancer slope factor (CSFo). When available, these toxicity values are taken from various USEPA sources. These sources, in order of preference for groundwater CTL development, are:

1) Integrated Risk Information System (IRIS).

2) Health Effects Assessment Summary Tables (HEAST).

3) National Center for Environmental Assessment (NCEA) provisional toxicity values.

4) Office of Pesticide Programs (OPP), *Reference Dose Tracking Report*; or Office of Water, *Drinking Water Regulations and Health Advisories*; or withdrawn values from IRIS or HEAST.

The last category consists of several sources of roughly equal preference.

When a toxicity value from NCEA is newer than, and appears to supercede, the toxicity value for that chemical in HEAST, the value from NCEA is used. Alternative approaches are used when toxicity values are not available from the USEPA. These alternative approaches include the use of "surrogate values," (i.e., toxicity values for substances from the same chemical class and with similar toxicological properties) and toxic equivalency factors (TEFs):

(1) ***Surrogate chemicals***

The use of toxicity values from chemically-related compounds offers a means to provide some estimate of risk, and of acceptable concentrations, for chemicals with little or no toxicity information. However, this approach carries with it significant uncertainty because small changes in chemical structure can produce profound differences in toxicity (compare CO and CO_2, acetate and fluoroacetate, ethanol and methanol, for example). The Table below lists the chemicals for which surrogate toxicity values are used in the development of CTLs presented in this report, the surrogate value, and the source of the surrogate value. It should be noted that all of the chemicals in question are considered non-carcinogens and therefore only surrogate oral reference doses are used.

Surrogate Toxicity Values

Contaminant	Surrogate Oral RfD (mg/kg-d)	Surrogate Contaminant
acenaphthylene	3.0E-02	pyrene[a]
benzo(g,h,i)perylene	3.0E-02	pyrene[a]
chlorophenol, 3-	5.0E-03	chlorophenol, 2-
chlorophenol, 4-	5.0E-03	chlorophenol, 2-
cymene, p	4.0E-03	butyltoluene, p-tert-
dichlorophenol, 2,3-	3.0E-03	dichlorophenol, 2,4-
dichlorophenol, 2,5-	3.0E-03	dichlorophenol, 2,4-
dichlorophenol, 2,6-	3.0E-03	dichlorophenol, 2,4-
dichlorophenol, 3,4-	3.0E-03	dichlorophenol, 2,4-
hexachlorocyclohexane, delta	3.0E-04	hexachlorocyclohexane, gamma
methylnaphthalene, 1-	3.0E-02	naphthalene
methylnaphthalene, 2-	3.0E-02	naphthalene
trichlorobenzene, 1,2,3-	1.0E-02	trichlorobenzene, 1,2,4-
trimethylbenzene, 1,2,3-	5.0E-02	trimethylbenzene, 1,2,4-

[a] For acenaphthylene and benzo(g,h,i)perylene, pyrene is chosen as a surrogate because its RfD is in the mid-range of RfDs for other non-carcinogenic PAHs. For all of the other contaminants in this table, the surrogate is chosen because it is the closest structurally-related compound with a RfD listed in IRIS.

(2) ***Toxic equivalency factors***

Toxicity equivalency factors (TEFs) are numerical expressions of the potencies of a series of compounds relative to the potency of a reference compound. For example, a chemical with a TEF of 0.5 would be only half as potent as the reference compound. Toxicity values for a series of compounds can be calculated using their TEFs and the toxicity value of the reference compound. For example, a provisional CSF for a chemical can be developed by multiplying its TEF by the CSF of the reference chemical. For non-cancer health effects, a provisional RfD can be derived by dividing the RfD of the

reference chemical by the TEF. In developing CTLs, the TEF approach is used to develop toxicity values for several carcinogenic polycyclic aromatic hydrocarbons (PAHs). In this case, benzo(a)pyrene is used as the reference chemical, and TEFs were obtained from the USEPA *Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons* (USEPA, 1993). For example, using this approach CSFs for dibenz(a,h)anthracene are developed using CSFs for benzo(a)pyrene and a TEF for dibenz(a,h)anthracene. TEFs are also available for other classes of chemicals; namely, the polychlorinated dibenzodioxins, the polychlorinated dibenzofurans (PCDFs), and polychlorinated biphenyls (PCBs). The use of the TEF values for these classes is somewhat different than for the PAHs. For these chemicals, the TEFs are used to express the concentrations of various chemicals in the class in terms of toxicologically equivalent concentrations of the reference chemical. For example, if a site has 1 ppm of 2,3,7,8-tetrachlorodibenzodioxin (TCDD, the reference chemical) and 4 ppm of a related congener with a TEF of 0.5, the total concentration in TCDD equivalents would be 3 ppm - (1 ppm x 1 for the reference chemical) + (4 ppm x 0.5) = 3 ppm. This concentration of TCDD equivalents would be compared with an CTL for dioxin based on TCDD, the reference chemical. A similar approach could be taken for PCBs, although analytical tests for PCBs rarely present the concentrations of individual PCB compounds. Usually, the total PCB concentration is compared with a toxicity value intended for the entire mixture of PCB compounds present.

For most chemicals, toxicity values used for the development of CTLs are available from USEPA sources whereas others had to be extrapolated using a combination of the above approaches. The identification of toxicity values needed for the derivation of CTLs primarily relied on surrogate values, route-to-route extrapolation, and the TEF approach. Extrapolation from occupational exposure limits, while useful, is only used to develop a CTL for a single contaminant (butyl alcohol, tert-). The toxicity values and their sources/bases are provided in Tables 5a and 5b.

III. Surface Water CTLs

A. Introduction

The surface water CTLs are equivalent to the numerical standards set forth in Section 24-11(4). Where such standards do not exist, the surface water CTLs are based on the following factors, as applicable: 1) numerical satandards set forth in Chapter 62-302, F.A.C.; 2) aquatic toxicity criteria; 3) human health risk calculations using a lifetime excess cancer risk level of one in a million (1×10^{-6}) and a hazard quotient of one (1) or less and 4) nuisance considerations. Therefore, the surface water standards presented herein are identical to those found in Chapter 62-777, F.A.C. except for those cases where numerical standards are specified in Section-12(11)(4) of the Code of Miami-Dade County (fluoride, iron and phenol). While some surface water CTLs are derived based on human health risk calculations and others are based on aquatic toxicity data, the goal is to provide surface water CTLs protective of both human health and the environment.

Chapter 24 surface water CTLs are listed in Table 1. For each surface water CTL, notation is provided indicating the source or basis for the derivation of that number.

B. Aquatic Toxicity Criteria

The method for deriving standards from aquatic toxicity information is borrowed from Chapter 62-777, F.A.C. as described in Figure 3A [Note: these are designated in Table 1 by the notation 'toxicity criteria' below the standard]. Generally, toxicity information from aquatic animals is used to calculate surface water CTL. In some circumstances, data from aquatic plants can also be used, as explained in Figure 3A. Basically, the procedure involves identifying the most sensitive relevant species and the median lethal concentration (LC_{50}) of the chemical in that species. The LC_{50} is then divided by 20 to obtain the surface water CTL.

C. Human Health Risk Equation

The equations used to derive a surface water CTL based on human health risk are shown in Figure 3B [Note: these are designated in Table 1 by the notation 'human health' below the standard]. There are separate equations for carcinogens and non-carcinogens. Both equations are based on partitioning of the contaminant from surface water to fish, and ingestion of the contaminated fish by humans. Examples of the calculation of a surface water CTL for a carcinogen and non-carcinogen are provided in Figure 3B.

1. Risk versus Hazard

The concepts of risk (as in estimating cancer risk or deriving cancer risk-based health criteria) and hazard (as in developing standards based on protection from non-cancer health effects) are as discussed in Section II.B.1. The target cancer risk used to calculate surface water CTLs for carcinogens is 1×10^{-6}. The target hazard quotient used in the equation to calculate surface water CTLs for non-carcinogens is 1.0.

2. Exposure Parameters

The equation for calculating surface water CTLs based on protection of human health is directed specifically to ingestion of contaminated fish. Critical exposure inputs in the equation include fish ingestion rate (assumed to be 6.5 g/day, per USEPA), body weight (70 kg), and a chemical-specific bioconcentration factor (BCF). The BCF represents the ratio of the concentration of the contaminant in fish to its concentration in surface water.

3. Toxicity Values

Toxicity values are taken from USEPA sources following the priority specified in Section II.B.3. The toxicity values used for derivation of surface water CTLs are included in Tables 5a and 5b, along with the sources of these values.

IV. Soil CTLs

A. Introduction

Soil CTLs are developed based on direct human contact (i.e., direct exposure), and based on soil serving as a source of groundwater contamination (i.e., leachability). In general, the methods and resulting values are similar to those in Chapter 62-777, F.A.C. The principal exceptions are leachability based on groundwater criteria soil CTLs, where differences in water CTLs have led to differences in acceptable concentrations in soils. These differences arise because of the changes introduced in the development of groundwater CTLs, specifically, the replacement of PQL values with health based standards, and the introduction of numerical standards specified for drinking water and surface waters in Sections 24-12(2)(H) and 24-11(4) of the Code of Miami-Dade County respectively.

B. CTLs for Direct Exposure to Soil

Direct exposure soil CTLs for carcinogens are calculated based on a target excess cancer risk of 1×10^{-6}. Direct exposure CTLs for non-carcinogens are derived using a hazard quotient of 1.0. All carcinogenic chemicals are also capable of producing non-cancer health effects, and CTLs based on non-cancer effects are calculated for carcinogens when suitable toxicity values are available. In nearly every case, the standard based on cancer risk is lower than the standard based on non-cancer effects, and the carcinogenicity-based value is used as the CTL. There are, however, a few exceptions. For example, the residential CTL for cadmium is based on the non-cancer endpoint because that value is lower than the CTL based on carcinogenicity.

1. Human Health Risk Equation

The equations used to calculate soil CTLs based on direct contact are presented in Figures 4 and 5. These equations are functionally equivalent to those used by USEPA Region IX to develop their preliminary remediation goals (USEPA, 1998). One equation is provided for calculating soil CTLs

based on non-cancer health effects and another for calculating standards based on cancer risk, as appropriate (i.e., if the chemical is regarded as a potential carcinogen). In the case of chemicals for which there is evidence of cancer and non-cancer health effects, the CTL is based on the more sensitive endpoint. Both the cancer and non-cancer equations consider three intake pathways from exposure to contaminated soils; 1) incidental soil ingestion, 2) dermal contact with contaminated soil, and 3) inhalation of chemicals volatilized from the soil or adhered to soil-derived suspended particulates. The combined and simultaneous impact of exposure from all three routes is used to calculate the soil CTLs. This is termed the *multi-route approach*, in contrast to the *route-specific approach* used by USEPA in their *Soil Screening Guidance* (SSG; USEPA, 1996b).

In the SSG, soil screening levels (SSLs) for a chemical are calculated separately for ingestion and inhalation exposure. In determining a soil screening level based on direct contact, the lower of the two values for a chemical would be selected. In most cases, intake through dermal contact is ignored unless there is evidence in the literature of substantial dermal absorption of the chemical (e.g., pentachlorophenol). In such instances, the SSL is adjusted to account for this uptake.

The main advantage of the multi-route approach is that it is more defensible on conceptual grounds. In the vast majority of possible scenarios, an individual will be exposed to contaminated soil through ingestion, dermal contact, and inhalation simultaneously. The multi-route approach considers the risk or hazard from a chemical to that individual to be the sum of the risks or hazards from each of these exposure routes. In contrast, the route-specific approach considers the risk or hazard posed by each route of exposure in isolation and makes the implicit assumption that risks or hazards from exposure to a chemical by multiple routes are unrelated, even if they involve the same target organ. Such an assumption could only be made if the toxicity posed by the chemical is route-dependent, which is seldom the case. For the vast majority of chemicals, the toxicity upon which the soil CTL is based is systemic in nature. That is, the reference doses and slope factors used to calculate the soil values are based on systemic toxicity endpoints, and a chemical reaching the target organ from any and all routes is

likely to contribute to toxicity. Under these circumstances it is difficult to consider the risks from the various routes of exposure to be less than additive.

In situations where risk from soil contamination is dominated by one exposure route, ignoring other routes has little effect on risk. Despite this small practical difference in soil levels between the multi-route and the route-specific approaches, the route-specific approach could conceivably result in compatibility problems with baseline risk assessments. This inconsistency cannot occur for CTLs developed using the multi-route approach since, like baseline risk assessments, they are based on risks summed from all relevant routes. In baseline risk assessments, the hazard index for a chemical is calculated from the sum of the hazard quotients for each of the exposure routes. When a CTL is based on exposure from only one of those routes, it provides an incomplete indication of hazard potential. To illustrate the potential problem, suppose a site with a chemical in the soil at a concentration just below its soil CTL developed using a route-specific approach. Because the concentration of the chemical is below the CTL, the risk assessor for the site might choose to drop it from the baseline risk assessment. If it is retained, however, its hazard index could be as high as 2. In this example, the use of a route-specific CTL can make possible the elimination from a baseline risk assessment of a chemical that would otherwise be flagged as posing a potentially unacceptable health risk. This inconsistency cannot occur for soil CTLs developed using the multi-route approach since, like baseline risk assessments, they are based on risks summed from all relevant routes.

a) Risk versus Hazard

The concepts of risk and hazard in the development of soil CTLs are the same as in the development of groundwater and surface water CTLs. Please refer to Section II.B.1 for a more complete discussion. The target cancer risk used in the calculation of direct exposure soil CTLs is 1×10^{-6}. The target hazard quotient for non-carcinogens is 1.0.

b) Exposure Parameters

Health risk evaluation of most sites can be conducted using CTLs based on either residential or industrial/commercial land use scenarios. Individuals potentially exposed in the case of residential land use include both children and adults, whereas only adults are assumed to be exposed to contaminated soil on sites designated for industrial/commercial use. [Note: For commercial uses involving significant regular soil contact by children, such as a school or day care facility, residential rather than industrial/commercial CTLs are applicable.]

Children are assumed to experience the greatest daily exposure to soil under residential land use scenarios. To be protective, residential CTLs must be based on childhood exposure assumptions in the case of chemicals for which risk is a function of their daily intake rate (as in the evaluation of non-cancer health effects). The exposure period for the evaluation of health risks that are a function of cumulative exposure (as in the evaluation of cancer risk) may include time spent both as a child and as an adult for the residential scenario. Physiological parameters such as body mass, surface area, and inhalation rate are obviously influenced by age. Parameters such as soil ingestion rate are also age-dependent. Time-weighted average values reflecting both childhood and adult exposures must be used in calculating CTLs for carcinogens applicable to residential land use. In this report, the individual exposed both as a child and as an adult is termed the *aggregate resident*.

Table 3 lists default exposure assumptions for both residential and commercial/industrial exposure scenarios used by USEPA to calculate default CTLs (i.e., CTLs applicable and protective for a broad range of sites). Some input parameters for the aggregate resident, such as inhalation rate and exposed dermal surface area, are not readily available from the USEPA and thus are developed from USEPA and NCHS (National Center for Health Statistics) data sources. The values calculated for these parameters are also listed in Table 3, and the method of derivation is described in Appendix A.

USEPA uses an age-adjusted soil ingestion rate of 114 mg-yr/kg-d in their SSG for the aggregate resident. This value is applied to a 30-year exposure period and is calculated by averaging a consumption of 200 mg of soil per day at a body mass of 15 kg for 6 years, with a consumption of 100 mg of soil per day at a body mass of 70 kg for 24 years (see USEPA, 1996b, for more information on the calculation of this value). There is a potential problem in using this approach because the dose-averaging is not directly comparable to that used to develop cancer slope factors. The typical approach to develop cancer slope factors is to use an average intake rate of the chemical divided by an average body mass over the exposure period, usually a lifetime in the case of rodent bioassays. To be strictly comparable, a similar approach should be used to derive the aggregate resident (time-weighted average) soil ingestion rate term. In developing Soil CTLs, dose-averaging for the aggregate resident is conducted in a manner consistent with that used in cancer slope factor development. Specifically, a time-weighted average soil ingestion rate of 120 mg/day is used, based on 6 years at 200 mg/day and 24 years at 100 mg/day. This is divided by a time-weighted average body mass of 59 kg, based on 6 years at 15 kg and 24 years at 70 kg. Although this averaging procedure produces values somewhat different from the USEPA, the values are technically more defensible.

The adherence factor (AF) represents the amount of soil that adheres to the skin per unit of surface area. Previously, the AF assumptions for residents and workers are taken from a range of values presented in USEPA's *Dermal Exposure Assessment: Principles and Applications* (USEPA, 1992). A different method of selecting the AF is used in calculating soil CTLs presented here, consistent with more recent USEPA guidance (RAGS Part E, USEPA, 2000). The newer approach is based on studies demonstrating that the amount of soil adhering to skin is different for different areas of the body. Additionally, data are now available regarding the soil loading that occurs on different regions of the skin during different activities. This information is used to derive weighted AF values for residents and workers, based on their anticipated activities and the areas of the body assumed to be exposed and available for soil contact. For example, as explained in Appendix A, the skin surface area assumed to be exposed for a child includes the head, forearms, hands, lower legs, and feet. Soil adherence data for these surfaces are averaged, weighting the contribution of the soil adherence for each part by its relative

surface area. [Note: Soil adherence data are available for the face only, rather than the entire head. In weighting the soil adherence data, adherence data for the face is conservatively assumed to be applicable to the entire head.] Adherence data are taken from the 95th percentile of observations of children playing at a daycare center, regarded as a typical (or central tendency) activity. The resulting weighted AF for a child resident (1 to 7 years of age) is **0.2 mg/cm^2**. The same weighted AF is obtained if soil adherence data from the 50th percentile is used for a high-contact activity (i.e., children playing in wet soil). For older children and adult residents, calculation of CTLs assumes that the head, forearms, hand, and lower legs are exposed. A different weighted AF is derived for these individuals, based both on different weighting from somewhat different surface areas exposed, as well as soil adherence data from different activities. In this case, soil adherence data from the 50th percentile of a high contact activity (gardening) is used to derive an AF of **0.07 mg/cm^2**. For workers, the head, forearms, hands, and lower legs are assumed to be exposed. Soil adherence data based on utility workers along with their respective surface areas are used to derive a weighted AF of **0.2 mg/cm^2** for the industrial/commercial worker scenario. Since the utility worker data are regarded as a high-end soil contact activity, the 50th percentile value is used. For the aggregate resident, the AF for the child (0.2 mg/cm^2) and the adult (0.07 mg/cm^2) are time-weighted to derive an average ([(6 years x 0.2)+(24 years x 0.07)]/30 years) of **0.1 mg/cm^2**.

One of the exposure variables, the particulate emission factor (PEF), is used to address intake from inhalation of contaminated soil-derived particulates. This value is a function of both site characteristics and local climatic conditions. The formula for calculating a PEF value is taken from the SSG (USEPA, 1996a) and appears in Figure 6. Default values from the SSG are used for the following parameters: vegetative cover, 50%; mean annual wind speed, 4.69 (m/s); equivalent threshold value of wind speed at 7 meters, 11.32 (m/s); and wind speed function F(x), 0.194 (unitless). The Q/C term accounts for the dispersion of contaminants from a source to the atmosphere and is dependent on the size of the area of contamination and local weather conditions. The USEPA SSG developed Q/C values for a variety of cities in the U.S. and contaminated sites of varying sizes. The Q/C value for a 0.5 acre contaminated site in Miami is selected for development of CTLs.

The volatilization factor (VF) is another input parameter used to assess the soil-to-air pathway of exposure. This term is used to define the relationship between the concentration of the chemical of concern in soil and its flux to air. The VF is calculated using an equation from the SSG as shown in Figure 7. Parameters related to characteristics of both the chemical and the soil are used in the calculation of a VF. Default soil characteristics specified in the SSG have been adopted for establishing default CTLs, although it is recognized that the relevant characteristics can vary widely among Miami-Dade County soils.

c) Chemical/Physical Properties

The equations for the calculation of CTLs for direct contact require the input of several chemical-specific values. These values are a function of the physical/chemical properties of each chemical of concern, and include the organic carbon normalized soil-water partition coefficient for organic compounds (K_{oc}), Henry's Law constant (HLC), diffusivity in air (D_i), and diffusivity in water (D_w). In some cases, it is necessary to calculate these values when published values do not exist. In this situation, additional physical/chemical values such as the density (d), water solubility (S), vapor pressure (VP) or the adsorption coefficient (K) are needed. In addition, the melting point (MP) is needed to assess the physical state of a chemical at ambient soil temperatures in order to determine the soil saturation limit (C_{sat}) for that chemical (see Section IV B.2 below). There are many sources for physical/chemical parameter values and, unfortunately, the values listed in various sources can differ dramatically. Therefore, it is important to have a designated hierarchy of sources for the selection of physical/chemical values to be consistent in the development of CTLs.

Chemical-specific values for MP, d, S, and HLC are preferentially selected from the *Superfund Chemical Data Matrix* (SCDM) (EPA/540/R-96/028), in agreement with the SSG. SCDM is accessible via the Internet and contains information selected from specified literature sources or other databases, and calculated values. The SCDM ranks the values that reasonably apply to a hazardous substance and reports a single value for each of the physical/chemical parameters. Values are taken

directly from the SCDM source tables rather than from the user interface because the source tables list several of the parameters to greater precision. The Hazardous Substance Data Bank (HSDB), The Agency for Toxic Substances and Disease Registry (ATSDR) *Toxicological Profiles*, or other reference texts (in that order of preference) are used when data for these parameters are unavailable from the SCDM. Values for d or HLC can be calculated using equations (1) and (2) below if they are not available from any of the sources mentioned above. The primary source for K_{oc} values is the SCDM. Secondarily, K_{oc} values are calculated from K_d values in the SCDM, using equation (3).

The primary source of diffusivity values is the CHEMDAT8 database (EPA/453/C-94/080B). If diffusivity values are not provided in the CHEMDAT8 database, they can be calculated using equations shown below taken from the literature accompanying the CHEMDAT8 database.

The following is the list of sources (in order of preference) for the chemical/physical parameters used in the development of the CTLs presented in this report.

<u>For HLC, d, S, VP, and MP</u>

1. The Superfund Chemical Data Matrix (SCDM)
2. The Hazardous Substances Data Bank (HSDB)
3. The Agency for Toxic Substances and Disease Registry's *Toxicological Profiles* (ATSDR)
4. Reference texts (e.g., CRC *Handbook of Chemistry and Physics* (Lide and Frederikse, 1994); *Handbook of Environmental Data on Organic Chemicals*, (Verschueren, 1996); *Handbook of Environmental Fate and Exposure Data for Organic Chemicals*, Volumes. I-V, (Howard, 1989, 1990, 1991, 1993, 1997); *Handbook of Physical Properties of Organic Chemicals* (Howard and Meylan,

1997); *Illustrated Handbook of Physical Chemical Properties and Environmental Fate for Organic Chemicals*, Volumes I-V (Mackay et al., 1992a,b, 1993, 1995, 1997).

5. Values calculated using equations from reference texts

For density (d):

$$d = \frac{MW}{5\sum_{i} n_i \bullet v_{a,i}} \quad (1)$$

where, MW = molecular weight of chemical (g/mol)

n_i = number of atoms i in a molecule

$v_{a,i}$ = relative volume of atom i (cm^3/mol)

Source: Baum (1998).

For Henry's Law constant (HLC):

$$HLC = \frac{VP \times MW}{S} \quad (2)$$

where, MW = molecular weight of chemical (g/mol)

VP = vapor pressure (atm)

S = solubility (mol/m^3)

Source: *Soil Screening Guidance: Technical Background Document* (USEPA, 1996b)

For K_{oc}:

1) Superfund Chemical Data Matrix (SCDM)

2) Calculated from the K_d published in SCDM using the following equation:

$$K_{oc} = K_d / 0.002 \quad (3)$$

3) The Hazardous Substances Data Bank (HSDB)

4) The Agency for Toxic Substances and Disease Registry's *Toxicological Profiles* (ATSDR)

5) Reference texts (see reference texts listed above)

Note: The K_{oc} and K_d parameters are used in the development of CTLs based on leaching to groundwater. In the case of some inorganic chemicals (antimony, arsenic, barium, beryllium, cadmium, chromium, mercury, selenium, silver, vanadium, and zinc), the SSG developed K_d's using the MINTEQ model and used them to generate soil-screening levels based on leaching to groundwater. For those chemicals, the SSG leachability value is cited in Table 2 rather than a value based on the K_d from SCDM.

For D_i and D_w:

1) The CHEMDAT8 database

2) Calculated using equations identified in the CHEMDAT8 database support document and shown below:

For diffusivity in air (D_i):

For compounds with a MW ≤ 100

$$D_i = 0.0067\ T^{1.5} \times (0.034 + MW^{-1})^{0.5} \times MW^{-0.17} \times [(MW / 2.5\ d)^{0.33} + 1.81]^{-2} \quad (4)$$

For compounds with a MW > 100

$$D_i = 0.0067\ T^{1.5} \times (0.034 + MW^{-1})^{0.5} \times MW^{-1.7} \times [(MW / 2.5\ d)^{0.33} + 1.81]^{2} \quad (5)$$

where, T = temperature, degrees Kelvin

MW = molecular weight of chemical (g/mol)

d = density of liquid chemical (g/cm^3)

For diffusivity in water (D_w):

$$D_w = 1.518 \times (10^{-4}) \times V_{cm}^{-0.6} \quad (6)$$

where, V_{cm} = molar volume of chemical (cm^3/mol)

Melting point (MP) is not available for all chemicals. If a specific MP could not be found in any of the reference sources, but a source listed it as a liquid, a default MP of –9.99 °C is assigned.

The precision with which the values from the various reference sources are reported can vary. In order to foster consistency in the development of CTLs, it is important to have a designated rounding policy for the physical/chemical values. Listed below is the precision to which values from reference sources are used in calculating the CTLs.

Input Precision for Physical/Chemical Parameters

Parameter	Numerical Precision
MW	2 decimal places
d	4 decimal places
HLC	3 significant figures
S	2 significant figures
MP	1 decimal place
VP	2 significant figures
K_{oc}	2 decimal places
D_i	3 significant figures
D_w	3 significant figures

The physical/chemical parameters for chemicals covered in this report are provided in Table 4.

The literature sources of physical/chemical values listed above are exhausted without finding a value for one or more of the required parameters for a few contaminants. For these cases, d and HLC values are calculated using the equations listed above. The tables below list the chemicals for which d and HLC values are calculated and the calculated values. For HLC, the VP values used in the calculations are also shown.

Calculated Density Values

Contaminant	Calculated Density
benomyl	1.2582
benzo(g,h,i)perylene	1.2683
chloro-m-cresol	1.2674
diuron	1.3320
heptachlor epoxide	1.5219
linuron	1.3588

Calculated Henry's Law Constants (HLC)

Contaminant	Vapor Pressure (mm Hg)	Vapor Pressure Source	Calculated HLC
chlorine cyanide	1.00E+03	Verschuren, 1996	9.51E-04
mercury, methyl	5.00E+01	HHS[a]	1.52E-02
zineb	8.00E-09	Howard, 1991	2.90E-09

[a] U.S. Department of Health and Human Services, Report on Alkyl Mercury

In addition, surrogate density values are used for nine chemicals. Surrogate density values are considered appropriate only when the density of an isomer of the chemical in question is available in the hierarchy of physical/chemical sources. The table below lists the chemicals for which surrogate density values are used, the value, and the source of the surrogate value.

Surrogate Density Values

Contaminant	Surrogate Density Value	Surrogate Contaminant
benzo(b)fluoranthene	1.3510	benzo(a)pyrene
benzo(k)fluoranthene	1.3510	benzo(a)pyrene
dichlorophenol, 2,3-	1.3830	dichlorophenol, 2,4-
dichlorophenol, 2,5-	1.3830	dichlorophenol, 2,4-
dichlorophenol, 2,6-	1.3830	dichlorophenol, 2,4-
dichlorophenol, 3,4-	1.3830	dichlorophenol, 2,4-
hexachlorocyclohexane, delta	1.8900	hexachlorocyclohexane, beta
indeno(1,2,3-cd)pyrene	1.3510	benzo(a)pyrene
phenylenediamine, p-	1.0096	phenylenediamine, m-

d) Toxicity Values

Toxicity values are preferentially obtained from USEPA sources. The hierarchy among USEPA sources is discussed in Section II.B.3, above. When toxicity values are not available from the USEPA, other approaches are used. Two of these approaches are the use of surrogate toxicity values and toxic equivalency factors. These are explained also in Section II.B.3. For development of direct exposure soil CTLs, one additional approach is needed — route-to-route extrapolation.

Direct exposure soil CTLs require toxicity values for ingestion, dermal contact, and inhalation routes of exposure for each chemical. Inhalation and dermal toxicity criteria are often not available. In these cases, route-to-route extrapolation is used to expand upon dose-toxicity relationships observed for one route of exposure to develop toxicity values for other routes. For example, the oral toxicity value can be used to derive corresponding inhalation or dermal values (see Appendix B). Intake from different routes is not necessarily equivalent, and information regarding toxicokinetics of the chemical (or

assumptions in this regard) must be taken into account when performing route-to-route extrapolation. Further, route-to-route extrapolation is not appropriate when there is evidence that the toxicity value serving as the basis for extrapolation is likely to be route-specific. If a slope factor (CSF) or a reference dose (RfD) is known or presumed to be route-specific, it is not regarded as suitable for route-to-route extrapolation. The toxicity values and their sources/basis are provided in Tables 5a and 5b.

2. Soil Saturation Limits

As described previously, the inhalation component of the CTLs for residential and industrial exposure to volatile contaminants is calculated using a VF. The equation for the VF (Figure 7) defines the relationship between the concentration of the chemical in soil and its flux to air, assuming an infinite source of the chemical and only vapor phase diffusion as the mechanism of transport. As emission flux increases, the air concentration increases, along with risks from inhalation exposure. The VF model assumes that this relationship holds throughout the possible range of chemical concentrations in soil, although at a sufficiently high concentration the soil pore air and pore water are saturated and the adsorptive limits of the soil particles are reached. Any increase in concentration beyond this point does not result in greater flux - the rate of flux reaches a plateau and volatile emissions (and air concentrations) can go no higher no matter how much additional chemical is present in soil. This concentration is termed the soil saturation limit (C_{sat}).

The C_{sat} value for a given chemical depends on many factors, including chemical-specific physical/chemical properties, as well as characteristics of the soil. As such, different chemicals present at a site will have different C_{sat} values, whereas the same chemical can have C_{sat} values that are different from site to site. A formula for estimating C_{sat}, using chemical-specific inputs and default soil assumptions, is shown in Figure 9.

Whenever the concentration of a chemical in soil exceeds its C_{sat} value, the standard formula for estimating volatilization and inhalation exposure will overestimate flux and inhalation exposure. This is

because it assumes that flux continues to increase with concentration. The failure of the VF model in recognizing the limit imposed by the saturation limit is of relevance in CTL development because the CTLs calculated for some chemicals (primarily volatile chemicals of low toxic potency) are greater than their C_{sat} values. This is the case for 23 of the chemicals for which CTLs are presented in this report.

Although it is possible to account for the influence of C_{sat} on the inhalation component of CTLs, it is decided to use the uncorrected CTLs, recognizing that this adds some extra measure of conservatism to the value. This approach is chosen mainly because accounting for C_{sat} requires that its value be estimated with some confidence for a wide variety of sites.

Although in theory C_{sat} can also influence the development of CTLs for leachability, in practice C_{sat} is not an issue of concern because nearly all leachability-based soil CTL s are well below their respective C_{sat}. Among the chemicals listed in Table 2, only di-n-octylphthalate and 1,1,2-trichloro-1,2,2-trifluoroethane have a leachability-based soil CTL that is greater than C_{sat}.

C_{sat} values may be useful for identifying situations in which free product is likely to be present. Soil concentrations above C_{sat} will indicate that the chemical may be present at the site as free product. It should be emphasized that the existence at a site of concentrations greater than the C_{sat} value does not necessarily signify that free product is present, but only that there is a distinct possibility that it is. As a site management tool for identifying the potential occurrence of free product (i.e., "red flagging"), C_{sat} values have been tabulated for a series of chemicals that can exist as liquids at room temperature. These are presented in Table 8. The actual occurrence of a chemical as free product at a given site must be determined by other means.

3. Acute Toxicity

Default residential direct exposure CTLs for non-carcinogenic chemicals are typically developed based on assumptions of chronic exposure, and are intended to be health protective for both children and adults. While it is generally assumed that these contaminant concentration limits are protective for

acute as well as chronic exposure, there may be circumstances where acute exposure is significantly larger than time-averaged chronic exposure. This larger exposure could result in acute toxicity.

A striking example of this situation can be seen with soil ingestion rates in children. While most children may ingest up to 200 mg of soil per day on average (the standard USEPA default assumption), in some instances episodic ingestion can be 250 times that amount or more. Wong et al. (1988) measured soil ingestion in children of normal mental capacity on one day per month for four months. He found that five of the 24 children ingested > 1 g on at least one of the four observation days, ranging from 3.8 to 60.7 g. Stanek and Calabrese (1995) used data from soil ingestion studies to develop a model to predict soil ingestion patterns in children. The results of this model indicated that "the majority (62%) of children will ingest > 1 g soil on 1-2 days/year, while 42% and 33% of children were estimated to ingest > 5 and > 10 g soil on 1-2 days/year, respectively." Although a soil ingestion rate of 5 g soil/day has been proposed by the USEPA (USEPA, 1986) to address the possibility that some children may exhibit soil pica (ingestion) in quantities far greater than the 200 mg/day value, this approach is regularly disregarded in practice. To prevent this oversight when assessing a site whose current or future uses may include contact with soil by small children, the potential for acute toxicity must be adequately addressed in the development of CTLs.

Calabrese et al. (1997) evaluated the potential for acute toxicity from a pica episode involving soil with contaminant concentrations regarded by the USEPA as conservative (i.e., at or below the USEPA Soil Screening Levels and USEPA Region III Risk-Based Soil Concentrations). Contaminant doses expected to result from a one-time soil pica episode of 5 to 50 g of soil were estimated and compared with acute doses demonstrated to produce toxicity in humans in poisoning episodes. The findings indicated that some residential soil cleanup targets levels could result, following a single large soil ingestion event, in doses in the range reported to produce acute toxicity, and even death. Of the thirteen chemicals included in the analysis, ingestion of soil containing cyanide, fluoride, phenol, or vanadium was found to result in a contaminant dose exceeding a reported acute human lethal dose. Ingestion of

barium, cadmium, copper, lead, or nickel from soil was found to produce doses associated with acute toxicity other than death.

Although the selective use of human data contributes greater confidence in the relevance and implications of these findings, it is important to acknowledge the limitations associated with this analysis. Estimates of the acute toxic and lethal doses were primarily extrapolated from reports on accidental ingestion, and exact dose estimation was difficult. In addition, most incidents of exposure were limited to adults; doses were then modified to approximate equivalent doses for children. Doses reported to produce toxicity in humans indicate only that the dose needed to cause the effect was met or exceeded; that is, they can only be used to approximate a LOAEL. For most of the effects of interest, data were insufficient to establish a NOAEL. Some case reports in the literature may represent sensitive individuals, and the extent to which dose-response information from these cases applies to the general population is uncertain. Also, the doses in this analysis were ingested doses rather than absorbed doses, and in many cases involved solutions from which absorption may be extensive. The presence of these contaminants in soil may reduce their bioavailability, and therefore their toxicity. Despite these limitations, the serious nature of acute toxicity potentially associated with consumption of contaminated soil during a soil pica episode requires that attention be paid to this issue when developing residential soil cleanup target levels.

The USEPA has acknowledged in the *Soil Screening Guidance: Technical Background Document* (USEPA, 1996b) that their residential screening values for cyanide and phenol may not be protective of small children in the event of acute soil ingestion episodes, but provides no guidance on how to address this problem.

The chemicals identified by Calabrese et al. (1997) as having the potential to produce an acute toxicity problem were evaluated to determine whether an adjustment in the residential soil CTL was required. Because the intake under these circumstances would be driven almost exclusively by ingestion, the CTL equation was altered to remove dermal contact and inhalation components. Also,

because the value is based on a single exposure event, terms related to averaging time and exposure frequency were deleted to produce the following equation:

$$CTL = \frac{BW}{\frac{1}{RfD_{acute}} \times SI \times CF}$$

where:

BW = body weight (kg)

RfD_{acute} = safe dose for acute exposure (mg/kg)

SI = amount of soil ingested (g)

CF = conversion factor for units (kg/g) (10^{-3})

Consistent with other CTLs based on exposure of a child, a body weight of 16.8 kg was assumed. So as not to make the derivation of acute toxicity SCTLs excessively conservative 10 g of soil was selected as the amount of soil ingested per event (SI), a value well within the range of observations reported by Calabrese and others (1997).

Unfortunately, safe doses intended specifically for acute exposures are not provided by the USEPA. An analysis was therefore required in order to develop RfD_{acute} values for each of the eight chemicals of interest — barium, cadmium, copper, cyanide, fluoride, nickel, phenol, and vanadium. The analysis focused primarily on studies and reports of poisonings in humans. For most of these chemicals, there is little in the way of acute toxicity studies in animals, and the studies that exist tend to focus on severe endpoints (e.g., death) and are of limited value in identifying lesser effects that still may be of concern. Also, the use of human data avoids the uncertainty in extrapolating observations across species.

The principal objective of the literature analysis was to identify the acute LOAEL or NOAEL for each chemical. Initially, this dose was then divided by an uncertainty factor (UF) and/or modifying

factor (MF) to produce a tentative acute toxicity reference dose (RfD_{acute}), analogous to the procedure used by the USEPA to develop chronic RfDs. UFs are intended to offer a safety margin in the face of uncertainty regarding extrapolation of doses (e.g., from animals to humans, from healthy subjects to sensitive subjects, etc.) and MFs can be applied to extend the safety margin when the database available for assessment is limited or weak. The calculated RfD_{acute} was then compared with the USEPA chronic oral RfD for that chemical or, in the case of copper, with dietary allowance guidelines. For many of the chemicals (e.g., cyanide), the calculated RfD_{acute} was lower than the USEPA chronic RfD for that chemical. This result represents an apparent conflict, since a dose that is safe to receive every day for a lifetime (i.e., the chronic RfD) should also be safe to receive on a single occasion. To avoid this conflict, the USEPA chronic RfD was adopted as the RfD_{acute} in these situations. Similarly, in the case of copper, application of any UF or MF other than 1 to an acute LOAEL resulted in a calculated RfD_{acute} lower than dietary allowance recommendations. As explained below (under "Copper"), the RfD_{acute} for copper was set at its upper limit for dietary intake in small children.

The appropriate doses representing the NOAEL or LOAEL for each chemical, as well as the appropriate UF and MF to be applied, were discussed by the Methodology Focus Group of the Contaminated Soils Forum, and in some cases modifications were recommended from values used in the previous, May 1999 technical support document. The values presented in this report reflect the recommendations of the Methodology Focus Group. As before, a distinction was made in the application of "safety factors" depending upon the toxic endpoint. Specifically, if the RfD_{acute} was based on transient gastrointestinal distress, a lower factor (UF and/or MF) was applied as compared with more serious toxic endpoints. This procedure reflects a risk management position that for acute soil ingestion, some risk of transient gastrointestinal distress is acceptable, but the CTLs should be fully protective against more serious toxicity (including more serious gastrointestinal effects).

A brief summary of the analysis for each of the eight chemicals appears below:

a) Barium

The toxicity of barium is very much dependent upon its water solubility. Barium sulfate, for example, is insoluble in water, is poorly absorbed, and is used safely in medicine as a radiocontrast medium. Soluble barium salts, however, are quite toxic and have been used as rodenticides. Numerous poisonings with soluble forms of barium have been reported in the medical literature. Some have resulted from accidental ingestion, suicide attempts, or mistaken use of a soluble form of barium for medical procedures (e.g., barium sulfide instead of barium sulfate). Perhaps the most significant reported incident of accidental poisoning with barium occurred when 144 persons ingested barium carbonate that was mistakenly substituted for potato starch in the preparation of sausage (Lewi et al., 1964; Ogen et al., 1967). Among the individuals poisoned, 19 were hospitalized and one died. Vomiting, abdominal pain and spasms, diarrhea, weakness, hypokalemia (decreased blood potassium levels), cardiac arrhythmias, paresthesias (abnormal sensation such as tingling), and muscle paralysis are typical signs and symptoms of barium poisoning (Ellenhorn et al., 1997). For barium carbonate, the lowest reported acute lethal dose is 57 mg/kg, and the lowest reported toxic dose is 29 mg/kg (Ellenhorn et al., 1997). Effects at this lowest toxic dose include flaccid paralysis, weakness, and paresthesia. Barium chloride appears to be somewhat more toxic. The lowest lethal dose is reported to be 11 mg/kg (Ellenhorn et al., 1997). McNally (1925) stated that "Kobert believes that under certain conditions, 2 g (barium) would be fatal. The toxic dose he believes to be 0.2 g." The latter value, which corresponds to about 3 mg/kg in a 70 kg adult, is similar to the threshold toxic dose of soluble barium compounds of 200-500 mg (i.e., 3-7 mg/kg), reported by the World Health Organization (WHO) (WHO, 1991). Unfortunately, the symptoms that constitute this reported threshold for toxic effects are unclear, and there is no clear distinction in the literature between doses that cause gastrointestinal symptoms and those producing more serious effects like paresthesia, muscle paralysis, and cardiac arrhythmia. The principal action of barium contributing to neuromuscular and cardiac toxicity is dysregulation of potassium. Experiments in dogs have found that an intravenous dose of 0.022 to 0.154 mg/kg produces significant decreases in serum potassium and the appearance of

abnormal electrocardiograms (Roza and Berman, 1971). This result suggests that the 3 mg/kg threshold dose applies equally to neuromuscular and cardiotoxicity, as well as to gastrointestinal effects.

Application of a UF of 100 (10 for sensitive subjects and 10 for extrapolation from a LOAEL to a NOAEL) to a LOAEL of 3.0 mg/kg yields a dose of 0.03 mg/kg. This value is lower than the current USEPA chronic oral RfD of 0.07 mg/kg-day. The value for the chronic oral RfD was therefore selected as the RfD_{acute}, resulting in an acute toxicity soil CTL for barium of 120 mg/kg.

b) Cadmium

With chronic exposure, the health effects of primary concern are renal toxicity and lung cancer. Both require long-term exposure, and neither is an issue with acute (one time) ingestion of cadmium. The health effects occurring at the lowest acute dosages are primarily gastrointestinal — nausea, vomiting, salivation, abdominal pain, cramps, and diarrhea (ATSDR, 1997a). Several cases of acute cadmium poisoning occurred during the 1940s and 1950s when cadmium was substituted for scarce chromium in plating cooking utensils and containers. In one report, two adults and four children experienced vomiting and cramps after drinking tea from a pitcher plated on the inside with cadmium (Frant and Kleeman, 1941). From information provided in their report, doses ranging from 0.2 to 1 mg/kg can be calculated. Other studies have reported that doses as low as 0.04 to 0.07 mg/kg cadmium are capable of inducing vomiting (Nordberg et al., 1973; and Lauwerys, 1979; as cited in ATSDR, 1997a). In all cases of cadmium ingestion within this dose range, recovery was rapid and complete, usually within 24 hours.

From these studies, it appears that the LOAEL for vomiting is about 0.05 mg/kg. Because the endpoint was gastrointestinal distress and the effect temporary, a UF and MF of 1 were applied. Using this value as the RfD_{acute}, an acute soil CTL of 84 mg/kg is calculated. This value is slightly higher than the residential CTL for cadmium based on chronic exposure (82 mg/kg), which was adopted as the residential CTL for cadmium to protect against toxicity from both acute and chronic exposure.

c) Copper

Several studies have reported that ingestion of drinking water or beverages with elevated copper concentrations results in gastrointestinal effects including nausea, vomiting, diarrhea, and abdominal pain (Knobeloch et al., 1994; Sidhu et al., 1995; ATSDR, 1990b). In fact, copper sulfate was used historically in medicine to induce vomiting (Goodman and Gillman, 1941). Three separate reports provide relatively consistent information regarding the doses of copper required to produce these effects. In one report, military nurses experienced nausea, vomiting, and diarrhea within 30 minutes to one hour after consuming cocktails from a copper-lined shaker (Wyllie, 1957). All but five of the 15 nurses experienced weakness, abdominal cramps, dizziness, and headache the next day. Reconstruction of the cocktail mixture and measurement of copper concentrations, coupled with consumption estimates for each of the nurses, can be used to derive copper dose estimates. The lowest dose (received by three of the nurses who became sick), was 0.09 mg/kg. Nicholas (1968) reported an incident in which 20 workmen became sick after drinking tea at work that contained 30 mg/L copper. All experienced nausea and several had diarrhea, with or without vomiting. The estimated dose of copper was 0.07 mg/kg. Spitalny et al. (1984) reported recurrent, acute gastrointestinal symptoms including nausea, vomiting, and abdominal pain in a family associated with drinking copper-contaminated well water, or beverages (juice or coffee) made with the water. Based on the concentration of copper in the water (7.8 mg/kg), a copper dose of 0.06 mg/kg is estimated. It is not clear whether children have increased sensitivity to gastrointestinal irritation from copper. One study of gastrointestinal complaints from copper in drinking water in two communities in Wisconsin found a greater prevalence of symptoms in children, but this difference could have resulted from higher exposures than adults (Knobeloch et al., 1994).

The acute gastrointestinal effects of copper in drinking water were investigated in a well-controlled prospective study (Pizarro et al., 1999). Sixty healthy adult women were randomly assigned drinking water containing 0, 1, 3, or 5 mg Cu/L for one-week intervals. During the study, the participants were reassigned into a different consumption group so that each individual received one

week of water at each of the exposure levels. At 3 mg/L Cu in water, a significant increase in gastrointestinal symptoms (nausea, abdominal pain, and vomiting) was reported. Using the mean water consumption (1.64 L/d) and body weight (63.6 kg) reported in the study, this result corresponds to a gastrointestinal effects dose of 0.077 mg/kg.

Copper is considered to be an essential element, and various recommendations for daily copper intake are only slightly below values shown to produce gastrointestinal distress. A WHO expert committee has recommended intake of 0.08 mg/kg-day for infants and children (as cited in NRC, 1989), and the American Academy of Pediatrics has recommended inclusion of copper in infant formulas that could result in approximately 0.4 mg copper per day (as cited in NRC, 1989). However, even while recognizing the nutritional importance of copper, health agencies caution against too much intake. A WHO/FAO guidance document - *Trace Elements in Human Nutrition and Health* (WHO, 1996) - discusses nutritional copper requirements in children and sets an upper limit of the safe range of copper intakes for children ages 1 to 6 years old of 0.09 mg/kg.

The best dose-response data for gastrointestinal distress from copper come from the study by Pizarro et al. (1999), and indicate a LOAEL of about 0.08 mg/kg. Application of a UF and MF of 1 (based on transient gastrointestinal distress as the endpoint) would yield a calculated RfD_{acute} of 0.08 mg/kg. Since this value is within dietary allowance limits for copper, the WHO-recommended copper intake limit of 0.09 mg/kg-day for small children was selected instead as the RfD_{acute}. This intake limit results in an acute toxicity residential soil CTL for copper of 150 mg/kg.

d) Cyanide

Cyanide is a potent and rapid-acting toxicant that has been involved in numerous intentional and accidental poisonings. The ATSDR reviewed the medical literature and determined that the average fatal dose of cyanide is 1.52 mg/kg (ATSDR, 1997b). The lowest human lethal dose reported in the medical literature is 0.56 mg/kg (Gettler and Baine, 1938). Comparisons of acute oral toxicity data

(with lethality as the endpoint) indicate that the toxicity of potassium cyanide, sodium cyanide, and hydrogen cyanide are similar on a molar basis. Symptoms of cyanide poisoning include anxiety, confusion, vertigo, and giddiness. Severe cases can result in loss of consciousness followed by convulsions, involuntary defecation, and death from respiratory failure (Gosselin et al., 1984). While clinical experience with cyanide is extensive, an upper-bound no-effect level has not been identified in humans. Any dose of cyanide capable of producing symptoms is potentially serious and medical attention will be required.

Clearly the best dose-toxicity information for cyanide exists for death as an endpoint, and when deriving an acute toxicity CTL for cyanide, the exceptional toxicity and steep dose-response curve of this chemical must be taken into consideration. There is no standard set of uncertainty factors to develop a safe dose based on a lethal dose, particularly one established in humans. Extrapolating from the average human lethal dose (approx. 1.5 mg/kg) places the safe acute dose below the USEPA chronic reference dose (0.02 mg/kg-day), even if a UF as small as 100 is used. There is little logic in placing the safe acute dose lower than the safe chronic dose used for risk calculations, and so the RfD_{acute} for cyanide was placed at a value equal to the USEPA chronic RfD. This procedure results in an acute toxicity CTL for cyanide of 34 mg/kg.

e) Fluoride

Because of the widespread use of fluoride compounds as supplements to municipal water supplies for the prevention of dental caries, there is substantial information available regarding the effects of fluoride in humans. Malfunctioning fluoridation equipment is often the cause of fluoride intoxications. In an elementary school, 34 children became ill from ingestion of over-fluorinated water (Hoffman et al., 1980). The intakes were estimated to range from 1.4 to 90 mg fluoride (based on a 20 kg body weight, which would result in an upper-end dose of 4.5 mg/kg). In another case, 22 adults become ill after ingesting water containing 1,041 mg/kg fluoride (Vogt et al., 1982). Doses producing nausea

alone were estimated at 1.2 mg/kg. More severe gastrointestinal symptoms were reported in those individuals who received doses of 2-3 mg/kg.

Fluoride supplements are often recommended for children who do not live in an area served by a fluorinated water supply. These tablets are typically flavored to aid in compliance and represent an important cause of accidental poisonings in the home. Spoerke et al. (1980) reviewed 150 reported cases of accidental poisonings with fluoride and found that a dose below 5 mg (absolute dose, not mg/kg) produced no gastrointestinal symptoms, a dose of 5-9 mg produced gastrointestinal symptoms in 10% of individuals, 10-19 mg caused symptoms in 21% of cases, 20-29 mg resulted in symptoms in 50% of cases, and 100% of individuals who ingested 30-39 mg were symptomatic. Augenstein et al. (1991) reviewed the medical records of children referred to the Rocky Mountain Poison Control Center for accidental fluoride ingestion. Of the 87 children included in the study, 70 had intake estimates sufficient to construct a dose response. Gastrointestinal symptoms predominated and included nausea, vomiting, diarrhea, abdominal pain and lethargy. Percentages of symptomatic patients, as a function of dose, were: < 1 mg/kg fluoride, 8%; 1-2 mg/kg fluoride, 17%; 2-3 mg/kg fluoride, 27%; 3-4 mg/kg fluoride, 50%; and 4-8.4 mg/kg fluoride, 100%.

Gastrointestinal symptoms from acute fluoride ingestion arise because fluoride is corrosive to the gastrointestinal tract. At higher doses, more severe toxicity can occur, including hypocalcemia, hyperkalemia, cardiac arrhythmias, muscle spasm, tetany, and convulsions (Spoerke et al., 1980; Augenstein et al., 1991)

Emergency medicine and toxicology texts often make recommendations about treatment options and dosages expected to produce serious adverse effects. Ellenhorn et al. (1997) suggested seeking immediate medical treatment for doses of fluoride exceeding 5 mg/kg. This is the same fluoride dose for which the CDC recommends prompt medical treatment (Reeves, 1995). Estimates of the lethal dose of fluoride in adults vary widely in the literature ranging from approximately 32 to 64 mg/kg. However, a 3-year-old weighing 12.5 kg died after ingesting 200 mg fluoride (16 mg/kg). The lowest reported

fatality from fluoride was in a boy of 27 months who died after ingestion of 50 mg of fluoride (Anonymous, 1979). Based on the mean body weight for his age (12 kg) the fatal dose was only 4 mg/kg. Two factors may have contributed to the severity of his reaction — the mother had been taking fluoride tablets during pregnancy and the child had received daily fluoride supplements (0.5 mg) for the 15 months prior to his death.

In developing a RfD_{acute} for fluoride, a 5 mg/kg dose was selected as the starting point. This is the dose above which clinical texts recommend seeking medical attention. Even though this guidance value is intended to be applicable to the general population, it was divided by a UF of 10 (for sensitive individuals) to yield a RfD_{acute} of 0.5 mg/kg. The acute toxicity soil CTL corresponding to this dose is 840 mg/kg. According to the study by Augenstein et al. (1991), the dose of fluoride in 10 g of soil at this concentration (0.5 mg/kg) would be expected to produce gastrointestinal symptoms in only a small percentage of children.

f) Nickel

There is only one report of death from acute ingestion of nickel. A 2-year old child ingested nickel sulfate crystals (570 mg/kg) and died from cardiac arrest eight hours later (Daldrup et al., 1986).

Sunderman et al. (1988) reported an incident in which 32 individuals drank from a water fountain contaminated with nickel sulfate and nickel chloride. It was estimated that the ingested doses ranged between 0.5 to 2.5 g of nickel. Twenty workers promptly developed symptoms of gastrointestinal distress including nausea, vomiting and abdominal cramps. Systemic effects included episodes of giddiness, lassitude, headache and cough. The lower end of the dose associated with adverse side effects was 7 mg/kg (assuming a 70 kg body weight).

The acute toxicity SCTL for nickel is based on a LOAEL of 7 mg/kg from the Sunderman et al. study. As with cadmium and copper, the toxic endpoint for the LOAEL is gastrointestinal effects.

However, unlike the gastrointestinal effects associated with the LOAEL for these other chemicals, the LOAEL for nickel came from a study in which 10 out of 20 of the poisoned individuals were hospitalized. Given this information, the LOAEL for nickel (unlike cadmium and copper) was divided by a UF of 10. It was also divided by an additional MF of 3, given the limited data upon which the LOAEL is based. This approach results in a RfD_{acute} of 0.2 mg/kg (0.23 rounded to one significant figure) for nickel. The corresponding soil CTL for nickel is 340 mg/kg.

In discussing the development of risk-based criteria for nickel in soils, it is worth noting that gastrointestinal effects are not the most sensitive effects of nickel. Nickel ingestion has been shown to produce dermal hypersensitivity reactions in individuals with nickel sensitivity. Nickel sensitivity appears to exist in about 10% of women and 1% of men. Nickel exposure in these individuals via the inhalation, dermal, or oral route results in dermal responses characterized by eczema, erythema, and dermal eruptions. Several clinical studies document the exacerbation of eczema and dermal eruptions following ingestion of nickel. Cronin et al. (1980) observed worsening of hand eczema in nickel-sensitive women from a single oral dose of as little as 0.6 mg nickel in solution. Studies by Jordan and King (1979) and Burrows et al. (1981) suggest that the NOAEL may be 0.5 mg nickel. Gawkrodger et al. (1986) reported that a single dose of nickel produced dermatitis, eczema, and measle-like eruptions on the limbs of women previously sensitized. All of the women responded to 5.6 mg, the dose they identified as the LOAEL from their study. Protection against dermal hypersensitivity reactions from nickel would require a RfD_{acute} lower than the current USEPA chronic oral RfD. In fact, the USEPA acknowledges in their IRIS record for nickel that the chronic oral RfD is probably adequate to prevent the development of nickel hypersensitivity, but may not protect nickel sensitive individuals from experiencing reactions at this dose.

g) Phenol

Acute ingestion of non-fatal doses of phenol results in burning mouth and gastrointestinal irritation and distress (Deichman, 1969). Bennett et al. (1950) reported an acute lethal dose of 230

mg/kg for an adult. Deichman (1969) reported the lethal range for adults to be between 14.3 mg/kg and 143 mg/kg. Interestingly, there is also a report of an ingestion of 14 mg/kg that caused only gastrointestinal effects (Cleland and Kingsbury, 1977). Intake of water contaminated with phenol for a period of several weeks resulted in diarrhea, burning mouth, and mouth sores (Baker et al., 1978). The dose calculated to have been ingested in these cases ranged from 0.14 to 3.4 mg/kg-day. Phenol is another chemical for which the USEPA acknowledges that their residential soil screening level based on chronic exposure may not be protective of children under acute exposure circumstances.

Application of a UF of 100 (10 for sensitive individuals and 10 for extrapolation from a LOAEL to a NOAEL) to the LOAEL for mouth lesions, 0.14 mg/kg-day, would yield a calculated RfD_{acute} of 0.0014 mg/kg, well below the USEPA chronic oral RfD of 0.6 mg/kg-day. The chronic oral RfD was therefore used as the RfD_{acute} value, resulting in a residential soil CTL of 1000 mg/kg.

h) Vanadium

Vanadium toxicity in humans primarily occurs following respiratory exposure in occupational settings, and data regarding toxicity following oral ingestion are lacking. However, vanadium has been examined for its therapeutic applications, including the treatment of syphilis, as a cholesterol-lowering agent (Dimond et al., 1963), and its ability to lower blood glucose in diabetic patients (Boden et al., 1996; Goldfine et al., 1985). Recently, vanadium supplements have been introduced to the consumer market for enhancing athletic performance (Fawcett et al., 1997).

From clinical studies, information is available regarding adverse side effects following oral ingestion of vanadium compounds. In several cases it was reported that patients experienced some form of gastrointestinal distress following oral ingestion of vanadium. Dimond et al. (1963) administered vanadium (ammonium vanadyl tartrate) to six patients for a period of six weeks. The subjects received 25, 50, 75 or 100 mg of the compound per day (0.36, 0.71, 1.1, and 1.4 mg/kg-day, assuming a 70 kg body weight). It is stated in the manuscript that all patients experienced gastrointestinal difficulties

manifested by diarrhea and cramps. Two patients reported greater fatigue and lethargy. The oral dosage for each patient was limited by cramping and diarrhea, and on a daily dosage of 50 mg or more, a purple-green tint developed on the tongue. Doses had to be lowered to 25 mg to reduce symptoms to tolerable levels.

In the study by Fawcett et al. (1996), two subjects receiving a 35 mg dose of vanadyl sulfate had to withdraw from the study due to health complaints. These studies collectively suggest that the threshold dose for gastrointestinal toxicity is probably close to 25 mg of these vanadium compounds. [Note: This value is very similar to the 30 mg/day dose of vanadyl sulfate commonly recommended as a dietary supplement.] Using the molecular composition of vanadyl sulfate, where vanadium comprises 31% of the total molecular weight, a 25 mg dose contains 7.8 mg vanadium. Assuming a 70 kg body weight for adults in these studies, this dose per unit body weight is 0.11 mg/kg. Since this endpoint is based on transient distress, a UF of 1 was applied. However, the LOAEL was divided by a modifying factor of 3 given the weakness in the data set available to assess toxicity, resulting in a RfD_{acute} of 0.04 mg/kg (rounded to one significant figure), corresponding to an acute toxicity soil CTL of 67 mg/kg vanadium in soil.

i) Summary of Residential CTLs Based on Acute Toxicity

The table below summarizes the RfD_{acute} values developed for each of the eight chemicals and the corresponding acute toxicity-based CTL. For comparison, the residential CTL for a child based on chronic exposure is also provided. The acute toxicity CTL is lower for each of the chemicals except cadmium. In all cases, the lower of the acute and chronic exposure-based CTL was adopted as the residential CTL. These values apply in situations where small children at play might come in contact with soils (e.g., residential areas, schools, daycare facilities, etc). They are not applicable for industrial sites.

Provisional Acute Oral Reference Doses and Corresponding Residential CTLs

Based on Acute Toxicity and Chronic Toxicity CTLs for Eight Chemicals

Chemical	Acute Oral Reference Dose (mg/kg)	CTL Based on Acute Toxicity (mg/kg)	CTL Based on Chronic Exposure (mg/kg)
Barium	7E-02	120	5800
Cadmium	5E-02	84	82
Copper	9E-02	150	3300
Cyanide	2E-02	34	1700
Fluoride	5E-01	840	5200
Nickel	2E-01	340	1600
Phenol	6E-01	1000	37000
Vanadium	4E-02	67	550

j) Caveats in the Acute Toxicity Analysis

There are several caveats in the acute toxicity analysis that should be acknowledged. These include the following:

- The focus of the analysis was intentionally on data relevant to acute (single dose) exposure in humans. In our opinion, these data are most pertinent in assessing potential human health risks from acute ingestion of soils. These data are limited, however, and there are several uncertainties inherent in human studies. Principal among these is the fact that doses must nearly always be estimated. The only alternative to this approach would be to use animal data. While dose estimation is more precise, studies of acute toxicity in animals are usually restricted to death as the endpoint, and extrapolation of safe human doses from lethal doses in animals is an extremely uncertain process.

- Despite efforts to update the analysis, the possibility remains that some poisoning reports or other relevant data were missed. In particular, studies appearing in the scientific literature during the first half of the century may be informative, but are very difficult to access because they cannot be identified through computerized search vehicles such as Medline and Toxline.

- The chemicals selected for this analysis were those identified by Calabrese et al. (1997) as representing a potential acute toxicity problem for children. While these are regarded as the most likely to pose an acute toxicity hazard, it is possible that there are other chemicals for which a similar concern is warranted. Should evidence arise that a chemical might pose an acute toxicity hazard for small children, the residential CTL for that chemical should be reconsidered.

- None of the studies in the analysis involved exposure to the chemical in soil. In most of the cases reported, the chemical was ingested in a soluble form, and the dose from soil required to produce equivalent toxicity may be much different. Presence of the chemical in soil in an insoluble form, or interactions between the chemical and soil that reduce its absorption from the gut could significantly reduce toxicity.

- A related issue deals with the form of the chemical. In some cases, the chemical can exist in more than one form, with substantial differences in toxic potential. Differences in bioavailability can contribute to these differences, but there can be other factors that influence the toxicity of different forms. Since default CTLs are intended to be applicable and protective, regardless of the form of the chemical, the choice in developing CTLs (including acute toxicity-based CTLs) has consistently been to use data from the most toxic form. It is recognized that this approach will overestimate risk in situations where a less toxic form is present.

C. Leachability-based Soil CTLs based in Groundwater Criteria

The migration of chemicals from soil to groundwater is modeled to identify chemical concentrations in soil that have the potential to result in contamination of groundwater (i.e., concentrations greater than the applicable water CTLs). Chemicals can migrate from soil to groundwater through a two-stage process that involves their release from soils into leachate and their transport to and within an underlying aquifer. Leachability-based soil CTLs are calculated using a standard linear equilibrium soil/water partition equation that estimates the release of chemicals of concern in soil leachate (USEPA, 1996b). In addition, a dilution factor is included to account for dilution of soil leachate in the aquifer and the leachability-based soil CTLs are then back-calculated from applicable water CTLs. In order to ensure protection of groundwater resources, groundwater leachability-based soil CTLs are calculated using the groundwater CTLs. Because contaminants may migrate to surface water via groundwater transport, surface water leachability-based soil CTLs are calculated using the freshwater and marine surface water CTLs. The applicable freshwater or marine surface water leachability- based soil CTLs are applicable when surface waters are, or are reasonably expected to be, impacted by contaminated groundwater.

1. Leachability Equation

In calculating the leachability-based soil CTLs intended for the protection of both groundwater and surface water bodies, the groundwater and surface water CTLs are first multiplied by a dilution attenuation factor (DAF) to derive a target leachate concentration. Soil characteristics that influence partitioning of the chemical between soil and water are also important components of the calculation. The complete equation for calculating CTLs based on migration of contaminants from soil to groundwater or surface is shown in Figure 8.

a) Input Parameters

The input of several chemical-specific factors is required in order to calculate CTLs based on leachability. These values include the organic carbon normalized soil-water partition coefficient for

organic compounds (K_{oc}) and the Henry's Law constant (HLC). The development of leachability-based soil CTL s for inorganic chemicals, however, requires the use of K_d values (soil-water partition coefficient). This is due to the fact that the relationship between soil organic carbon content and soil sorption is not as robust for inorganic (metals) as it is for organic chemicals. In addition, it is sometimes necessary to calculate other physical/chemical values such as density (d), water solubility (S), vapor pressure (VP), or adsorption coefficients (K). Since different references for physical/chemical parameters can cite very dissimilar values, a hierarchy of sources for these values is recommended as outlined in Section IV.B.1.c (above). Chemical-specific values for d, S, VP, and HLC are generally selected from the Superfund Chemical Data Matrix (SCDM) (EPA/540/R-96/028). K_{oc} values are primarily obtained from the SCDM. Secondarily, K_{oc} values are calculated from K_d values in the SCDM using the equation $K_{oc} = K_d/0.002$. In cases where data are not available from the SCDM, the Hazardous Substance Database (HSDB), ATSDR Toxicological Profiles, or other reference texts (in that order of preference) are used.

b) Leachability of Metals

The estimation of leachability-based soil CTLs values for most metals is more difficult than for organic chemicals. Unlike organic compounds, K_d values (soil/water partition coefficient) for metals are significantly affected by a variety of soil parameters. This has prompted DERM to recommend the use of a laboratory leaching procedure instead of the soil/water partition equation for some of the metals (e.g., aluminum, cobalt, copper, fluoride, iron, lead) [Note: these are designated in Table 2 by the footnote '***']. Site-specific leachability-based soil CTLs for metals derived using K_d values estimated using the MINTEQA2 model are considered acceptable, but only if oily wastes are not present.

c) Laboratory Leaching Procedure

The Synthetic Precipitation Leaching Procedure (SPLP), developed to model an acid rain leaching environment, can be used when there are no oily soil chemicals of concern and the decision is

made to determine site-specific leachate values. Testing should involve a minimum of three representative soil samples, pursuant to USEPA Test Method 1312 (SPLP). Leachate concentrations from SPLP should not exceed the applicable water CTL (e.g. groundwater, freshwater or marine surface water). SPLP should not be used for contaminants derived from used oil or similar petroleum products as the presence of these products may interfere with the quantification of the sample result. When leachability testing of samples containing oily wastes is required, the Toxicity Characteristic Leaching Procedure (TCLP) should be used.

D. Site-Specific Soil CTLs

Default soil CTLs are useful tools during site evaluation and for the formulation of remediation strategies for a broad range of sites. However, there will be some sites in which site-specific characteristics are different from the assumptions used to calculate default CTLs such that these CTLs do not accurately correspond to the risk goals for that site. This section identifies variables in the CTL equations for which site-specific information can be substituted in order to obtain more accurate CTLs, as well as some considerations in making site-specific modifications.

1. Site-Specific Soil Characteristics

Site soil characteristics can influence the rate of volatilization of organic chemicals into air, and thus the level of chemical in soil that is acceptable in terms of direct contact. Soil characteristics are also important determinants of leaching potential for chemicals from soils to groundwater or surface water.

The measurement of appropriate soil characteristics to develop site-specific VFs may be useful, particularly if risks from soil at a site are thought to be dominated by inhalation of volatile chemicals. Parameters necessary for the determination of the VF include the average soil moisture content (ω), the dry soil bulk density (ρ_b), fraction of organic carbon (f_{oc}), and soil pH, which is used to select pH-specific K_{oc} and K_d values. Methods for determining these site-specific measured values for the derivation of the VF are listed below and outlined in the SSG (USEPA, 1996a).

Methods and Data Sources Used to Calculate VF

Soil Characteristic	Data Source	Method
Soil moisture content (ω)	Lab measurement	ASTM D 2216
Dry soil bulk density (ρ_b)	Field measurement	All soils: ASTM D 2937; shallow soils: ASTM D 1556, ASTM D 2167, ASTM D 2922
Soil organic carbon (f_{oc})	Lab measurement	Nelson & Sommers (1982)
Soil texture	Lab measurement	Particle size analysis (Gee and Bauder, 1986) and USDA classification; used to estimate θ_w & I
Soil pH	Field measurement	McLean (1982)

The following table provides the equations, sources, and methods for deriving soil characteristics using site-specific data.

Derivation of Site-Specific Soil Characteristics

Soil Characteristic	Data Source	Method
Water-filled soil porosity (θ_w) (Average soil moisture content)	$\theta_w = \eta\ (I/K_s)^{1/(2b+3)}$ or $\theta_w = \omega\rho_b$	Where, η = total soil porosity (L_{pore}/L_{soil}) I = infiltration rate (m/yr) K_s = saturated hydraulic conductivity (m/yr) b = soil-specific exponential parameter (unitless) ω = soil moisture content (g_{water}/g_{soil}) ρ_b = dry soil bulk density (g/cm^3)
Total soil porosity (η)	$\eta = 1 - (\rho_b/\rho_s)$	Where, ρ_b = dry soil bulk density (g/cm^3) ρ_s = soil particle density = 2.65 kg/L
Infiltration rate (I)	HELP model; Regional estimates	HELP (Schroeder et al., 1984); may be used for site-specific infiltration estimates; used to calculate θ_w
Soil-specific exponential parameter (b) (Moisture retention component)	Look-up	Attachment A (USEPA, 1996a); used to calculate θ_w
Saturated hydraulic conductivity (K_s)	Look-up	Attachment A (USEPA, 1996a); used to calculate θ_w
Air-filled soil porosity (θ_a)	$\theta_a = n - \omega\rho_b$ or $\theta_a = n - \theta_w$	Where, n = total soil porosity (L_{pore}/L_{soil}) ω = soil moisture content (g_{water}/g_{soil}) ρ_b = dry soil bulk density (g/cm^3) θ_w = average soil moisture content (L_{water}/L_{soil})
Soil-water organic partition coefficient (organics) (K_d)	$K_d = K_{oc} \times f_{oc}$	Where, K_{oc} = chemical-specific soil-organic carbon partition coefficient (cm^3/g) f_{oc} = organic carbon content of soil (g/g)

The use of default CTLs may be more cost-effective and less time consuming than developing site-specific values, because site-specific data on soil characteristics must be collected over a one-year period. In addition to the time needed for the collection of site-specific data, the investigator must be in

strict accordance with the approved methods. This condition is particularly important because the collected data are used for the derivation of several site-specific parameters. Therefore, errors in the collection of one set of data would result in several incorrectly derived values. For example, θ_w and θ_a are derived from the soil moisture content (ω). To adequately generate ω, the soil moisture content must represent the *annual* average. Discrete soil samples may be affected by preceding rainfall events, and thus they would not accurately represent the moisture content, resulting in the incorrect derivation of θ_w and θ_a. Correctly deriving values such as θ_a is of great significance because, other than the initial soil concentration, air-filled soil porosity (θ_a) is the most significant soil parameter affecting the volatilization of chemicals of concern from soil. A higher θ_a will signify a greater potential for emission of volatile chemicals of concern from soil.

The development of site-specific leachability-based soil CTLs may be useful because soil characteristics at a given site may bear little resemblance to the default assumptions. It should be recognized that site-specific CTLs for leachability that are calculated based on the equation presented in Figure 8 could be either higher or lower than the default standards because the default assumptions are not skewed toward the conservative end of the range of values possible for Miami-Dade County. Site-specific characteristics that are important when calculating a leachability-based soil CTL include the f_{oc}, θ_w, θ_a, η, and ρ_b. Additional information on developing site-specific input values are described in the SSG (USEPA, 1996a).

2. TRPH Speciation

The TRPH CTLs are developed to be used in a two-tiered approach, as described in detail in Appendix C, with a primary TRPH soil CTL as the starting value. Primary TRPH soil CTLs for direct exposure and leachability included in Table 2 are based on the assumption that the TRPHs consist exclusively of aromatic hydrocarbons in the $>C_8$-C_{10} range. While CTLs derived for hydrocarbons in the C_5-C_7 range are the most restrictive (Table C4, Appendix C), these compounds are not detected using the Florida Petroleum Residual Organic (FL-PRO) analysis. Currently, the FL-PRO method of TRPH analysis is limited to measuring the concentration of mixed petroleum hydrocarbons in the range

of C_8-C_{40}. While FL-PRO does not measure hydrocarbons in the C_5-C_7 range, the most toxic and prevalent chemicals among these are addressed by other analyses and individual CTLs. Therefore, the primary TRPH CTL is based on the most conservative and health protective carbon range that can be detected by FL-PRO, the >C_8-C_{10} carbon range.

TRPH CTLs are derived from chemical/physical parameters and toxicity values assigned to each carbon range as described in Appendix C. It should be noted, however, that while the >C_8-C_{10} aromatic fraction has the most restrictive inhalation RfD, the >C_{16} aromatic fractions currently have the most restrictive oral RfD (TPHCWG, 1997b; Table C3, Appendix C). Therefore, under certain site-specific conditions in which there may be elevated soil moisture and fraction organic carbon, such that volatilization would not be a significant consideration relative to ingestion, the potential exists for the >C_{16} aromatic hydrocarbon concentrations to pose the greater risk.

If the primary CTL is exceeded, it is proposed that a second tier would be employed, such that each TRPH sub-classification would possess its own CTL. However, individual CTLs could not be set for each C-range because the current FL-PRO method of analysis cannot distinguish between aliphatics and aromatics. Additionally, the quantitation of individual compounds is difficult and not confirmative, as only "fresh" petroleum hydrocarbons provide distinct peaks in analysis by gas chromatography (GC). Weathered petroleum hydrocarbons such as those found at contaminated sites, produce unresolved complex mixtures ("hills"), not peaks when analyzed by GC. Therefore, one can only obtain an estimate over the entire C-range of the fraction of petroleum hydrocarbons that are present in the sample. Furthermore, it is possible that the human health CTL for TRPH, with relatively low toxicity and low mobility potential, could result in staining, odors and other nuisance conditions. As such, the CTL may not address all of the potential issues of concern, depending upon the setting and management of a particular site.

3. Other Site Characteristics

The volatilization (VF) is a function of local climatic conditions and the size of contaminated area as expressed in the Q/C term. The USEPA (1996b) has tabulated Q/C values for contaminated areas ranging from 0.5 to 30 acres in size for selected cities around the U.S., including Miami. These values are based on a modeling exercise that incorporated, among other things, meteorological data for these cities. The default Q/C recommended in Figure 7 is based on Miami data and a 0.5 acre contaminated area. A site-specific Q/C term should be considered if the area of contaminated soil is significantly greater than 0.5 acres and inhalation exposure is a significant concern.

The VF equation assumes an infinite mass for the source of the chemical of concern. The VF equation can be modified to take mass of chemicals of concern into consideration when the volume of contaminated soil is known (i.e., the area and depth). An alternative VF equation incorporating estimates of volume of contaminated soil is described in the SSG (USEPA, 1996a, 1996b).

Although the VF model used in this report is capable of adjusting the VF for different durations of exposure, it is limited to exposures that begin immediately. The model assumes that the rate of flux of a volatile chemical from soil to air is highest when the concentration in surface soil is highest and that it declines over time, so that the air concentration of the chemical also declines over time. Consequently, everything else being equal, the average concentration in air will depend on the averaging period (or exposure duration) such that longer periods will have lower average concentrations. This is because, as the concentration in soil declines over time, lower concentrations are included in the averaging process. For example, the model predicts that, for a given concentration of xylene in soil, the average concentration over the first six years will be approximately twice the average concentration over the first 25 years because the air concentrations in later years are quite low. In some site-specific situations, other exposure periods may be relevant, including exposures that do not begin immediately. In these circumstances, a useful approach may be the use of the EMSOFT program, developed by the USEPA National Center for Environmental Assessment. VFs calculated by EMSOFT do not differ from those calculated with the current VF model for exposure durations that begin immediately. However,

EMSOFT will compute average soil VFs for exposure intervals beginning and ending at any time in the future. Therefore, EMSOFT may be of value in deriving site-specific volatilization factors for exposure scenarios that differ from default assumptions.

Local wind conditions can influence the Particulate Emission Factor (PEF) and could conceivably be used to adjust the PEF in the development of site-specific CTLs. Because PEF is a quantitatively important factor in the CTLs of only a very few chemicals, there is generally little incentive for developing site-specific PEF values. It is important to note that the PEF is applicable only for undisturbed soil. If there is significant soil disturbance at a site, such as from vehicular traffic, site-specific estimates of dust levels may have to be substituted for the PEF in deriving CTLs.

The dilution attenuation factor (DAF) is a parameter that is important when calculating leachability-based soil CTLs. The USEPA selected a default DAF using results from the EPACMTP Model. This model uses a Monte Carlo analysis with input parameters obtained from nationwide surveys of waste sites and from applying the soil screening level dilution model to 300 groundwater sites across the country. The model distributions were repeated 15,000 times for each scenario and a cumulative frequency distribution of DAF values was generated. Results from an accompanying sensitivity analysis indicated that climate, soil type, and size of the contaminated area have the greatest effect on the DAF. To gain further information on the national range and distribution of DAF values, the dilution model was applied to two large surveys of hydrogeologic site investigations. These surveys are the American Petroleum Institute's hydrogeologic database (HGDB) and the USEPA's database of conditions at DNAPL sites. DAF modeling information from a total of 300 sites indicated that the geometric mean DAF of all sites combined was 20 for a source area of 0.5 acre. This value was carefully selected using a "weight of evidence" approach which best represents a nationwide average and is therefore regarded as an acceptable default for use at most sites. For sites with contamination greater than 0.5 acre, it may be necessary to derive a site-specific DAF. The aquifer hydraulic conductivity, the hydraulic gradient, the mixing zone depth, the infiltration rate, and the source length

parallel to groundwater flow must be determined in order to derive a site-specific DAF (USEPA, 1996a).

E. Special Cases

1. Ammonia

Ammonia is an inorganic compound that exists in a state of equilibrium between un-ionized ammonia (NH_3) and ammonium ion (NH_4^+). The state of ionization, and thus the percentages present as NH_3 and NH_4^+ are generally dependent upon the pH of the medium (i.e., soil or water), and to a lesser degree upon temperature. Higher pH results in a greater percentage as NH_3, whereas lower pH favors the formation of NH_4^+.

Some environmental criteria are intended to be applied to NH_3 specifically, while others are applied to total ammonia (NH_3 plus $NH4^+$). For this reason, ammonia is listed twice in Tables 1 and 2, with the designation of "Ammonia (as total)" applicable to NH_3 plus $NH4^+$, and the designation of "Ammonia" applicable solely to the pH dependent NH_3 content of the sample. For example, the groundwater CTL for ammonia (as total) of 2800 µg/L is applicable to the sum of the NH_3 and NH_4^+ concentrations. Alternatively, the surface water CTL for ammonia in freshwater of 20 µg/L, taken from Chapter 62-302, F.A.C., is for NH_3 only, and compliance must be determined based on estimated NH_3 levels. Since standard analytical methods only provide information on total ammonia concentration, the concentration of NH_3 in samples must be estimated based on the total ammonia concentration and the pH of the water. Marine and freshwater surface water CTLs also exist for total ammonia (500 µg/L). In this situation, analytical data for total ammonia should be applied without modification to determine compliance.

Site-specific soil characteristics may greatly affect the ionization of ammonia and therefore the potential for leaching. Leachability is based, in part, on the partitioning of a compound between soil and water. For organic contaminants, the partitioning is dependent on the organic carbon normalized

partitioning coefficient (K_{oc}). However, the simple relationship between soil organic carbon and sorption observed for organic compounds does not apply to inorganic contaminants such as ammonia. The soil-water distribution constant (K_d) for inorganic compounds is affected by numerous geochemical parameters and processes, including pH, sorption to clays, organic matter, iron oxides, other soil constituents, oxidation/reduction conditions, major ion chemistry, and the chemical form of the inorganic present. The number of significant influencing parameters, and their variability among sites within Miami-Dade county may contribute to differences in K_d values of several orders of magnitude with similar variability in the resulting leachability based on groundwater criteria soil CTLs. For sites where ammonia leachability is a concern, leachability based on groundwater criteria soil CTLs may require site-specific adjustments or SPLP analyses may be required, to reflect leachabilities particular to site-specific conditions.

Two sets of leachability based on groundwater criteria soil CTLs are provided for ammonia in Table 2. One set of leachability values is based on surface water CTLs for ammonia as NH_3; another set is derived using total ammonia groundwater and surface water CTLs. Leachability-based CTLs are calculated according to the equations in Figure 8 using the default parameters listed and the appropriate chemical-physical constants listed in Table 4.

Direct exposure soil CTLs for ammonia (as total) are derived using the default equation for non-carcinogens (see Figure 5) and an oral reference dose of 0.4 mg/kg-day, based on a minimal risk level (MRL) derived by ATSDR (ATSDR, 1990a)[1]. For the inhalation route of exposure, an inhalation reference dose of 0.03 mg/kg-day is used. This dose is derived from the inhalation reference

1 The oral MRL for ammonia currently listed in the ATSDR Toxicological Profile for Ammonia is 0.3 mg/kg-day. This value was derived by adjusting the NOAEL of 40 mg/kg/day by an uncertainty factor of 100 and an adjustment factor for intermittent exposure. Per discussion with John Wheeler at ATSDR it was indicated that the use of an intermittent exposure factor in the extrapolation of the NOAEL to the MRL is no longer recommended. As such, the ATSDR recommended oral MRL for ammonia has been modified to 0.4 mg/kg/day and the drinking water MRL is 14,000 µg/L. Although an MRL of 14 mg/L exists for ammonia in drinking water, a value of 2800 µg/L was used here since it incorporates a relative source contribution factor of 20%, which FDEP includes in the development of groundwater guidance concentrations for non-carcinogens.

concentration of 0.1 mg/m^3 (IRIS). Given that the percentage of total ammonia present as NH_3 depends on soil pH, direct exposure soil CTLs are conservatively developed by assuming that all of the ammonia in soil is in the NH_3 form. This is because, while ammonia as NH_3 has a significant capacity to volatilize, NH_4^+ does not and it will be fully dissolved in water within the soil matrix. Consequently, for ammonia in soil, ingestion exposure is not as important as inhalation because once ingested the potential toxicity of NH_3 and NH_4^+ will be similar due to equilibrium between the two forms in the presence of gastric acids. When volatilization is minimal (i.e. low soil pH, see table below), the direct exposure soil CTL will be driven primarily by the oral component. The ammonia CTLs that are based on oral and dermal exposure pathways only are 35,000 mg/kg and 870,000 mg/kg for residential and industrial scenarios, respectively. Alternatively, at higher soil pH, the CTL for ammonia is predominantly driven by the inhalation component of the equation, and therefore reflects the capacity of these compounds to volatilize. In these cases, the inhalation component of the CTL equation must be adjusted to account for the proportion of ammonia available for volatilization. Thus, to accurately select a direct exposure soil CTL for ammonia on a site-specific basis, the soil pH must be known. Otherwise one must conservatively assume that 100% of ammonia is present as NH_3 (i.e. the assumption built in the default CTL). The table below provides CTLs for ammonia based on soil pH at an ambient soil temperature of 25°C.

Range of CTLs for Direct Exposure to Soil (at 25° C)

Soil pH[a]	Percent Un-Ionized Ammonia (NH_3)[b]	Residential (mg/kg)[c]	Industrial (mg/kg)[c]
	100%	**750**	**4000**
9.5	64.3%	1200	6200
8.5	15.2%	4400	26000
7.5	1.77%	19000	180000
6.5	0.18%	32000	630000
6.0	0.0568%	34000	780000
5.5	0.0180%	35000	840000
5.04[d]	0.00624%	35000	860000
5.0	0.00569%	35000	870000

[a] Increasing ammonia concentrations will tend to increase soil pH. Situations in which pH of soils is low and ammonia concentrations are high are unlikely to exist at contaminated sites.

[b] USEPA: Aqueous Ammonia Equilibrium-Tabulation of Percent Un-Ionized Ammonia, EPA/600/3-79/091.

[c] Calculated by dividing 550 mg/kg or 37000 mg/kg by the percent corresponding to the selected pH, but limited by the oral route contribution (35000 mg/kg residential and 870000 mg/kg industrial).
[d] Average pH of Florida soils.

2. Lead

a) Residential

The residential direct exposure soil CTL for lead is based on the Office of Solid Waste and Emergency Response (OSWER) Directive #9355.4–12 *Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Correction Action Facilities* (USEPA, 1994a). The guidance level for lead in soils described in this directive is calculated with the USEPA's *Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children* (USEPA, 1994b). This model accounts for the multimedia nature of lead exposure in children and calculates distributions of exposure and risk likely to occur at a site using default assumptions. Young children are particularly sensitive to the effects of lead and require specific attention in the development of a CTL for this heavy metal. Thus, a soil CTL that is protective for young children is also expected to be protective for older persons. The 400 mg/kg guidance level for lead in residential soils cited in the 1994 OSWER directive is calculated such that a hypothetical child would have an estimated risk of no more than 5% of exceeding the 10 µg/dL blood lead concentration. This blood lead level is based on research conducted by the Centers for Disease Control and USEPA that associates blood lead levels exceeding 10 µg/dL with health effects in children.

b) Industrial

The approach outlined in *Recommendations of the Technical Review Workgroup for Lead for an Interim Approach to Assessing Risks Associated with Adult Exposures to Lead in Soil* (USEPA, 1996d) (TRW) is followed to calculate the industrial direct exposure soil CTLs for lead. This guidance document provides methodology for assessing risks associated with non-residential adult exposures to lead in soil based on the most sensitive worker population, i.e. women of child-bearing age. The methodology focuses on estimating fetal blood lead concentrations in pregnant women that are

exposed to lead contaminated soil. That is, the model is designed to estimate an acceptable soil lead concentration to which women could be exposed, while pregnant, without the risk of producing unacceptable blood lead concentrations in the developing fetus, i.e., blood levels above 10 µg/dL.

This method is based on a simplified representation of lead biokinetics assumed to predict quasi-steady state blood lead concentrations among adults (women of child-bearing age) who are relatively consistently exposed to a lead-contaminated site. In addition, a constant of proportionality between fetal blood lead concentration at birth and maternal blood lead concentration is also employed. As such, this model provides a means for consistency in calculating acceptable industrial soil lead levels.

A series of equations, discussed in detail in the TRW document, are used to derive an acceptable lead concentration in soil. $PbB_{adult,central,goal,}$ is derived first. This value represents the risk-based goal for the central estimate of blood lead concentrations in adult women that ensures the fetal blood lead concentration goal of 10 µg/dL is not exceeded. This value is derived from the following equation:

$$PbB_{a,c,g} = \frac{PbB_{fetal,0.95,goal}}{GSD_{i,adult}^{1.645} \times R_{fetal/maternal}} \qquad (4)$$

where,

$PbB_{fetal,0.95,goal}$ = 10 is the goal for the 95th percentile blood lead concentration (µg/dL) among fetuses born to women having exposures to the specified site soil concentration.

R = 0.9 is the constant of proportionality between fetal blood lead concentration at birth and maternal blood lead concentration.

GSD is the geometric standard deviation for blood lead concentrations among adults having exposures to similar on-site lead concentrations but having

non-uniform response to site lead (intake, biokinetics) and non-uniform off-site lead exposures.

Ideally the GSD used in the model is estimated from the population of concern at the site. In the absence of site-specific blood lead data, the TRW recommends a GSD range of 1.8-2.1 µg/dL based on an evaluation of blood lead concentration data for different types of populations. For homogeneous populations, a value of 1.8 is recommended whereas 2.1 is recommended for heterogeneous populations. For the default industrial direct exposure soil CTL, heterogeneity of populations at a workplace is assumed. Thus, the GSD selected from the recommended defaults is 2.1 µg/dL, resulting in a $PbB_{a,c,g}$ = 3.28 µg/dL.

Next, the target blood lead concentration ($PbB_{a,c,g}$) is employed along with other variables to calculate PbS, which represents the CTL.

$$PbS = \frac{(PbB_{a,c,g} - PbB_{a,0}) \times AT}{BKSF \times IR_{soil} \times AF_{soil} \times EF_{soil}} \quad (5)$$

where,

$PbB_{a,c,g\ (adult,\ central,\ goal)}$ = 3.28 – 4.23 µg/dL

$PbB_{a,0\ (adult,\ background)}$ = 1.7 – 2.2 µg/dL

AT = 365 days/year

BKSF (biokinetic slope factor) = 0.4 µg/dL per µg/day

IR_{soil} (ingestion rate) = 0.05 g/day

AF_{soil} (absorption factor) = 0.12 [unitless]

EF_{soil} (exposure frequency) = 219 days/year

In this equation, the baseline blood lead concentration, $PbB_{a,0}$, represents the adult blood lead concentration (µg/dL) in the absence of site exposures. This value is intended to be the best estimate of a reasonable central value of blood lead concentration in women of childbearing age who are not exposed to lead-contaminated non-residential soil or dust at the site. Ideally, this value is obtained from

a representative sample of adult women from the area. In the absence of site-specific data, the TRW recommends a range of 1.7-2.2 µg/dL, which is representative of women between the ages of 20 and 49 years. For Chapter 24, an average value of 1.95 µg/dL is selected, taken from the middle of the range of values provided by the TRW. In the TRW model, the baseline $PbB_{a,0}$ is subtracted from the target $PbB_{a,c,g}$ to obtain a value representative of the allowable increase in blood lead level that will not cause an exceedance of the target blood lead level. This value corresponds to 1.33 µg/dL (3.28 µg/dL minus 1.95 µg/dL) using the default values selected for Chapter 62-777, F.A.C. Additionally, the model uses an averaging time of 365 days/year, an exposure frequency of 219 days/year (based on USEPA guidance for average time spent at work by both full-time and part-time workers), and an exposure duration of one year (not shown in the denominator of the equation because it is 1). The other variables are defined as follows:

• BKSF = Biokinetic slope factor. Increase in the typical adult blood lead concentration in relation to increases in average daily lead uptake. Recommended value is 0.4 µg/dL blood lead increase per µg/day lead uptake.

• AF_{soil} = Fraction of lead in soil ingested that is absorbed from the gastrointestinal tract. TRW recommends a default value of 0.12 based on the assumption that the absorption factor for soluble lead is 0.2 and that the relative bioavailability of lead in soil compared to soluble lead is 0.6, thus 0.2 x 0.6 = 0.12.

• IR_{soil} = Intake rate of soil. Recommended value is 0.05 g/day.

Although this default value addresses all occupational soil intake by an individual (whether directly from soil or indirectly through contact with dust) risks associated with more intensive soil contact activities such as construction and excavation are not included. Thus, site-specific data on soil contact intensity should be considered when evaluating the applicability of the default industrial direct exposure soil CTL. Larger ingestion rates may be more

appropriate in some cases, depending on the duration and type of exposure scenario being evaluated.

Using equations (4) and (5) with the recommended defaults and values selected to best represent a contaminated site, it is possible to calculate the CTL for lead as follows:

$$PbB_{a,c,g} = \frac{10\mu g/dL}{2.1^{1.645} \times 0.9} = 3.28\mu g/dL$$

CTL for lead =

$$\frac{(3.28\mu g/dL - 1.95\mu g/dL) \times 365 days/yr}{0.4\mu g/dL \text{ per } \mu g/day \times 0.05 g/day \times 0.12 \times 219 days/yr} = 923.6 \text{ or } 920 mg/kg$$

A value of 920 mg/kg lead is calculated as the industrial direct exposure soil CTL. Applying other default values provided in the TRW model documentation results in lead soil CTLs that range from 750 mg/kg to 1800 mg/kg. A soil lead value within this range can be derived on a site-specific basis following the guidance in the TRW document for selection of appropriate default values based on population statistics and descriptions, and provided the soil intake rate is 0.05 g/day.

Although the TRW approach recognizes that more detailed blood lead kinetics models could provide better estimates in cases of brief acute exposures or intermittent exposure patterns, the methodology provided by the TRW is the recommended approach pending further development and evaluation of other biokinetic models.

3. Methyl mercury

Most USEPA-approved analytical methods for determining methyl mercury concentrations in soil are based on measurements of total organic mercury. Soil concentrations reported as methyl mercury

may, however, include other organic mercury species. Recognizing this, default CTLs for methyl mercury are developed in a way that would be protective for organic mercury species in general. Data regarding the comparative toxicity of organic mercurial compounds is limited, and the USEPA has developed a RfD_o only for methyl mercury. Although this value is tentatively assumed to be applicable to all forms of organic mercury, the physical/chemical properties of organic mercury compounds can vary significantly. For example, dimethyl mercury is much more volatile than methyl mercury, making the dose received from a given concentration in soil much higher. Consequently, the physical/chemical properties of dimethyl mercury are used to derive the default methyl mercury CTL to ensure it is also protective under circumstances of dimethyl mercury exposure. Alternative standards for specific forms of this heavy metal (including methyl mercury) can be utilized when employing analytical methodologies capable of reliably speciating organic mercury.

4. Polychlorinated Dibenzodioxins (PCDDs) and Polychlorinated Dibenzofurans (PCDFs)

Polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PDCFs) are typically found in the environment as mixtures of PCDD or PCDF congeners. The individual PCDD and PCDF congeners can vary widely in terms of toxic potency, and mixtures with different congener composition, but the same total concentration, can therefore pose different risks. Most analyses of PCDDs and PCDFs in environmental samples provide information on the congeners present. The current approach to assessing the toxicity of these mixtures involves the use of toxic equivalency factors (or TEFs), which are discussed in Section II.B.4 and in the *Interim Procedures for Estimating Risks Associated with Exposures to Mixtures of Chlorinated Dibenzo-p-Dioxins and –Dibenzofurans (CDDs and CDFs) and 1989 Update* (USEPA, 1989b). For dioxin-contaminated sites, concentrations of dioxin congeners should be converted to 2,3,7,8-TCDD equivalents using the most current recommended TEFs. **The total dioxin concentration**, in 2,3,7,8-TCDD equivalents, should then be compared with the dioxin CTL. The dioxin CTL is also applicable to PCDFs, given the similarity in the toxicity of these two classes of chemicals. For sites with PCDF contamination, PCDF concentrations should be converted to 2,3,7,8-TCDD equivalents using the most current recommended

TEFs. The total PCDF concentration, in 2,3,7,8-TCDD equivalents, is then compared with the dioxin CTL. For sites with both PCDF and PCDD contamination the sum of the TEQs for both classes of compounds should be compared to the CTL for 2,3,7,8-TCDD. [Note: TEFs are also available to convert PCB congeners to 2,3,7,8-TCDD equivalents. However, USEPA currently recommends evaluating risks from PCBs separately through the use of PCB-specific reference doses and cancer slope factors.]

V. Toxicological Interactions Among Chemicals

Exposure to combinations of chemicals may result in interactions leading to a significant increase or decrease in the overall toxicity of the mixture compared to the summation of the toxicity of the individual chemicals. As a result, the concept of toxic interactions from multiple chemical exposures is a subject of considerable interest and concern for hazardous waste sites where multiple chemical exposures are probable.

Interactions among toxic compounds may occur through an alteration in the absorption, distribution, metabolism, and excretion of one chemical by another, which will lead to a change in its toxicity. Animal studies have demonstrated the occurrence of such interactions among gaseous pollutants, pesticides, metals, and solvents. Interactions may also occur when one chemical alters the responsiveness of cells and target organs to the effects of other chemicals, such as through receptor up-regulation or altered cell-signaling pathways. Very little information exists on toxic interactions in humans, and inferences must be made from studies of toxicant effects in laboratory animals. Care must be exercised when evaluating these data because, even in circumstances where significant interactions have been observed, 1) the dosages at which the interaction occurs are usually not well characterized; 2) there is often uncertainty as to whether the mechanism for the interaction is relevant to humans, particularly at the comparatively low levels of exposure typically encountered from contaminated environmental media; and 3) most such studies involve exposure to two chemicals, whereas exposure at contaminated sites can involve several toxicants. Due to the limitations mentioned above, the utility of

these observations in evaluating the human health implications of multiple chemical exposures is limited, and it is extremely difficult to address chemical interactions in quantitative risk assessment other than on a rather simplistic level.

In baseline risk assessments for contaminated sites the standard approach is to assume that risks to the individual from multiple chemicals of concern are, at most, additive. The incremental excess cancer risk to the exposed individual is the sum of the cancer risks from individual carcinogenic chemicals of concern present at the site. Hazard quotients for individual non-carcinogenic chemicals are summed only when there is evidence that the chemicals may have additive effects. If two chemicals share the same mechanism of action or the same target organ for toxicity, their effects are usually considered potentially additive.

The initial assessment of risk (and hazard) posed by site contaminants, as part of a tiered site evaluation, requires an approach that is both relatively simple and conservative. These objectives can be achieved for most sites by assuming that risks posed by the contaminants present are additive. In the case of cancer risk, it is recognized that the cancer risks from individual chemicals are not truly independent, and therefore some error will be introduced in calculating total cancer risk from the sum of the individual cancer risks. However, since the probability of developing cancer from environmental exposure to contaminants is usually small, the error in summing them will also be small and of little consequence in estimating total cancer risk. When more than one carcinogen is present at a site, the direct exposure CTLs in Table 2 must be adjusted to reflect total cancer risk. To ensure that the total cancer risk does not exceed one-in-a-million (1×10^{-6}), CTLs from Table 2 for each carcinogen should, when appropriate, be divided by the total number of carcinogens present at the site to derive site-specific CTLs.

In the case of non-carcinogens, additivity of effect is most likely to occur when the contaminants affect the same target organ. With this concept in mind, initial evaluation of a site should, when appropriate, employ CTLs adjusted to reflect additivity in target organ toxicity. For contaminants

affecting the same target organ, CTLs from Table 2 for each should, when appropriate, be divided by the number of contaminants affecting that organ. For example, if four contaminants present at a site characteristically produce liver toxicity, the relevant CTLs for these chemicals would be their direct exposure CTL values in Table 2 divided by four. To assist in identifying chemicals affecting the same target organ, Table 5b lists each of the non-carcinogenic chemicals of concern for which a CTL is derived for this report, the reference dose for that chemical, and the toxic endpoint upon which the reference dose is based. To further facilitate the identification of chemicals with common target organs and/or effects, Table 6 lists the chemicals sorted by target organ or effect.

If risks are unevenly distributed among chemicals at a site, the simple method of apportionment described above for deriving site-specific CTLs may lead to sites where total risk is below the goals of 1×10^{-6} and a hazard index of 1. In these circumstances, a weighted approach for calculating CTLs may be more appropriate. For example, consider the situation of four chemicals that affect the same target organ, each with an CTL of 1 ppm. Chemical A is present at 0.05 ppm, Chemical B at 0.1 ppm, Chemical C at 0.25 ppm, and Chemical D at 0.9 ppm. Since there are four chemicals present that affect the same target organ, the CTL for each would be divided by 4 — in this case leading to an CTL of 0.25 ppm for each. In this example, only chemical D poses a potential problem (i.e., it is present at a concentration greater than its modified CTL of 0.25 ppm). Cleanup of Chemical D to its CTL of 0.25 ppm would lead to a total hazard index of only 0.65 for all four chemicals. If a weighted apportionment is used instead, Chemical D could be cleaned to 0.55 instead of 0.25 ppm, and still retain a hazard index < 1.

Although in principle some interactions among chemicals may result in greater-than-additive effects, at present there are no specific examples indicating that the additive approach described above is not sufficiently conservative for initial site evaluation purposes. If evidence arises in the future for specific interactions that would render this approach less than health-protective, the approach should be modified to take these interactions into consideration.

The need for more detailed, site-specific risk assessments may require the incorporation of quantitative information on toxicological interactions as a means to specifically evaluate the potential for additivity. However, simple additivity is the most commonly recommended approach for risk assessment.

Additivity may result from *dose addition*, which occurs when chemicals act on similar biological systems and elicit a common response, whereas *response addition* occurs when chemicals act by independent mechanisms to produce toxicity of the same organ or tissue (Hertzberg et al., 1997). With *dose addition*, the chemicals are assumed to be functional clones following similar pathways of uptake, metabolism, distribution and elimination, and eliciting the same toxic effect. Thus, although the dose of one chemical may be too small to elicit an effect, the addition of a second chemical may be enough so as to increase the total dose to a level that results in an adverse effect. Under *response addition*, different physiologic pathways are followed and the response to one chemical occurs whether or not the second chemical is present. For example, the liver may be the common target organ, but the mechanism of injury can differ (e.g., peroxisomal proliferation, induction of oxidant stress, protein adduction). However, it is the sum of the responses at the common target organ that is measured as the additive effect, regardless of the differences in mechanism of action. *Dose addition* should always be treated as a summation of hazard quotients. *Response addition*, however, may not always be accurately characterized by a simple summation of hazard quotients, depending upon the toxic mechanisms involved. In cases of *response addition*, approaches other than simple addition can be used to derive site-specific CTLs, but must be carefully justified by the mechanism(s) of action of the chemicals and supported by empirical observations.

Other chemical interactions, such as antagonism, inhibition, masking, synergism, and potentiation should be considered in the context of a detailed, site-specific risk assessment. As with *response addition*, manipulation of CTLs based on these interactions should be soundly and carefully based on mechanistic principles supported by empirical observations from the peer-reviewed scientific literature.

VI. Caveats and Uncertainties

There are several caveats to the CTLs developed here. First, CTLs for direct contact with soils are based on protection of human health. Some of them may not be protective of other species. For example, direct exposure soil CTLs for some metals (e.g., beryllium) exceed concentrations proven to be toxic to some plant species (USEPA, 1996b). Second, it should be recognized that the CTL methodology presented in this report does not include human health risk that may occur through indirect intake pathways such as uptake into plants and animals that are used as food. However, intake of site contaminants from food sources is not regarded as a major exposure pathway in most situations. For special circumstances where individuals make extensive use of crops or animals grown on contaminated soils, these CTLs may not be appropriate.

CTLs may not be sufficiently protective for some sites. There may be situations in which exposure exceeds the default assumptions employed in developing default CTLs, e.g., workers with extensive soil contact and opportunity for exposure, such as construction workers involved in excavation, or children with soil pica. Finally, the CTLs methodology does not address other important issues such as objectionable odors and visible staining of various media.

VII. References

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3. HALs: Drinking Water Regulations and Health Advisories (EPA/822/B-96/002)
   http://www.epa.gov/OST/Tools/dwstds1.html

4. HSDB: Hazardous Substance Database
   http://www.nlm.nih.gov/pubs/factsheets/hsdbfs.html
   (This address provides information on how to subscribe to HSDB.)

5. IRIS: Integrated Risk Information System
   http://www.epa.gov/ordntrnt/ORD/dbases/iris/index.html

6. MINTEQA2: Metal Speciation Equilibrium for Surface and Groundwater
   http://www.epa.gov/earth100/records/a00164.html

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IX. List of Acronyms and Definitions

Acute Exposure:	A single, brief exposure, usually less than 24 hours in duration.
Acute Toxicity:	The ability of a substance to cause adverse health effects from an acute exposure.
Antagonism:	When toxic effects from exposure to a combination of chemicals are less than what is expected based on their individual toxicities.
Aliphatic Hydrocarbon:	A chemical composed of hydrogen and carbon in which the carbon atoms form a chain.
Aromatic Hydrocarbon:	A chemical composed of hydrogen and carbon that contains one or more aromatic (benzene) rings.
ATSDR:	Agency for Toxic Substances and Disease Registry
BCF:	Bioconcentration Factor. The ratio of the concentration of a contaminant in a given organism to its concentration in the surrounding medium (water, soil, etc.).
Bioavailability:	The rate and extent of systemic absorption of a chemical.
CAS number:	A unique identification number assigned to a chemical by the Chemical Abstract Service.
CERCLA:	Comprehensive Environmental Response, Compensation, and Liability Act
Chronic Exposure:	Repeated or continuous exposure occurring over an extended period.
Chronic Toxicity:	The ability of a substance to cause adverse health effects as a result of chronic exposure.
Cleanup:	Actions taken to deal with a release or threat of release of a hazardous substance that could affect human and environmental health. The term cleanup is sometimes used interchangeably with the terms remedial action, removal action, response action, or corrective action.
Contaminant:	Any undesired physical, chemical, biological, or radiological substance that is present in the air, water, or soil.

C_{sat}:	Soil saturation limit. The concentration in soil at which the absorptive limits of the soil particles, the solubility limits of the soil pore water, and saturation of soil pore air have been reached.
CSF:	Cancer Slope Factor. A dose-response metric derived from human or animal studies that is used to calculate cancer risk.
CTL:	Cleanup Target Level: Enforceable numerical value that set limits on the amount of contaminants present in various media.
DAF:	Dilution Attenuation Factor. The numerical factor by which contaminant concentration is diminished as it moves through soil and groundwater from its source to the point of contact. As chemicals leach from soil and move through groundwater, attenuating effects include adsorption of the contaminant onto soil and aquifer media, chemical transformation, biological degradation, and dilution from mixing of leachate with ambient groundwater.
Default CTLs:	CTLs for soil, surface waters and groundwater that are applicable and protective for a broad range of sites.
DERM:	Miami-Dade County Department of Environmental Resources Management
Dermal Absorption:	The process by which a chemical penetrates the skin and enters the systemic circulation.
Detection Limit:	The lowest concentration of a chemical that can be distinguished from zero or background.
Dermal Exposure:	Contact between a chemical and the skin.
Dermal Toxicity:	Adverse effects of a toxicant on the skin.
Dose:	The quantity of a chemical administered to an organism or to which it is exposed. The absorbed dose is the amount that is absorbed and enters the body.
Exposure:	In the context of this report, exposure refers to contact with a toxicant.
Exposure Route:	The route by which a toxicant enters the body — through the lungs (from inhalation), through the skin (from dermal contact); or through the gastrointestinal tract (from ingestion).
FDEP:	Florida Department of Environmental Protection

FL-PRO:	Florida Petroleum Residual Organic analysis
Free Product:	A contaminant present in environmental media in a pure or undissolved state, usually as a liquid.
GC:	Gas Chromatography. An analytical technique for detecting and quantitating chemicals. This technique uses an instrument called a gas chromatograph.
GSD:	Geometric Standard Deviation
Hazard:	Potential for a chemical to produce adverse health effects.
HEAST:	USEPA Health Effects Assessment Summary Tables
HGDB:	American Petroleum Institute's Hydrogeologic Database
HLC:	Henry's Law constant
HQ:	Hazard Quotient. The ratio of the projected dose of a chemical resulting from exposure divided by the appropriate reference dose for that chemical.
HSDB:	Hazardous Substances Data Bank
IEUBK:	Integrated Exposure Uptake Biokinetic Model. A model developed by the USEPA to predict blood lead concentrations in children resulting from exposure to lead in soil and other sources.
Inhibition:	When the toxic effect of a chemical is reduced by the presence of a second substance that does not have that toxic effect.
IRIS:	Integrated Risk Information System. A USEPA electronic database containing toxicity values (e.g., reference doses and slope factors).
ISF:	Inhalation Slope Factor. A dose-response metric based on human or animal studies that is used to calculate cancer risk from inhalation exposure.
IUR:	Inhalation Unit Risk. A chemical-specific value that, when multiplied by the concentration of the chemical in air, yields the excess cancer risk associated with that concentration.
LC_{50}:	Median Lethal Concentration. The concentration of a toxicant that is lethal to 50 percent of the test organisms within a designated period.

LD_{50}:	Median Lethal Dose. The dose of toxicant that is lethal to 50 percent of the test organisms within a designated period.
LOAEL:	Lowest Observable Adverse Effect Level. The lowest dose of a chemical observed to cause an adverse effect.
Leaching:	The process by which soluble constituents are dissolved from, and transported through, the soils by water.
Masking:	When concurrent toxic effects of two or more chemicals are opposite or functionally competing, reducing or obscuring their individual toxic effects.
MRL:	Minimal Risk Level. A safe dose (or dosing rate) for a chemical developed by the Agency for Toxic Substances and Disease Registry, U.S. Public Health Service.
NCEA:	USEPA National Center for Environmental Assessment
NCHS:	National Center for Health Statistics
NHANES:	National Health and Nutrition Survey
NOAEL:	No Observable Adverse Effect Level. The highest dose of a chemical observed not to produce an adverse health effect.
NRC:	National Research Council
OPP:	USEPA Office of Pesticide Programs
Organoleptic:	Based on taste or odor.
OSHA:	Occupational Safety and Health Act. Sets minimum health and safety standards for the workplace.
OSWER:	USEPA Office of Solid Waste and Emergency Response
PAH:	Polycyclic Aromatic Hydrocarbon
PCB:	Polychlorinated Biphenyl
PCDF:	Polychlorinated Dibenzofuran
PEF:	Particulate Emission Factor. A term used to relate the concentration of a contaminant in soil with its concentration in air as dust particles. Factors that are used to determine the PEF include the extent of dust dispersion,

	the extent of vegetative cover, wind speed, and the extent to which the soil surface is erodible.
Porosity:	Degree to which soil, gravel, sediment, or rock is permeated with pores or cavities through which water or air can move.
Potentiation:	When the toxic effect of a substance is increased by the presence of a second chemical that does not have that toxic effect.
PQL:	Practical Quantitation Limit. A concentration below which quantitation is unreliable.
Q/C:	Technically, the inverse mean concentration at the center of a square source. When calculating the concentration of volatiles or dust in the air, it is the term that represents their dispersion in the atmosphere. Q/C values are derived from air modeling and can vary depending upon climatic conditions and the size of the contaminated area.
RCRA:	Resource Conservation and Recovery Act
Remediation:	Cleanup or other methods used to remove or contain a toxic spill or hazardous materials from a contaminated site.
RfC:	Reference Concentration. An estimate of the concentration of a toxicant that is likely to be without appreciable risk of adverse effects during a lifetime of continuous exposure.
RfD:	Reference Dose. An estimate of the dose of a toxicant that, when given every day over a lifetime, is likely to be without appreciable risk of adverse effects. The RfD is specific for the route of exposure (i.e., ingestion versus dermal versus inhalation).
Risk:	A measure of the probability that an adverse effect will occur in exposed individuals or the environment as a result of a specified exposure.
Route of Exposure:	The route by which a chemical comes into contact with an organism, e.g., inhalation, ingestion, or dermal contact.
RSC:	Relative Source Contribution. The fraction of the total allowable intake of a chemical allocated to a particular source (such as intake of contaminated groundwater).
SCDM:	Superfund Chemical Data Matrix

SPLP:	Synthetic Precipitation Leaching Procedure. A method for predicting leaching of a chemical from soil to water under typical environmental conditions.
SSG:	Soil Screening Guidance. A USEPA document describing the development of soil screening levels (SSLs).
SSL:	Soil Screening Levels. Risk-based screening levels for chemicals in soil developed by the USEPA.
Surrogate:	A substance that shares similar chemical and/or physical properties with another substance. When toxicity or physical/chemical properties for a chemical are unavailable, values from another, surrogate chemical may be used in the development of its CTL.
Synergism:	When the toxic effect from exposure to two or more chemicals is greater than what is expected based on their individual toxicities (i.e., the effects are greater than additive).
TCDD:	Tetrachlorodibenzo-*p*-dioxin. Sometimes refers to 2,3,7,8-tetrachloro-*p*-dibenzodioxin, which is the most toxic congener.
TCLP:	Toxicity Characteristic Leaching Procedure. A method for predicting leaching of a chemical from soil to water under conditions that might exist in a landfill.
TEFs:	Toxic Equivalency Factors. Numerical expression of the potencies of a series of related compounds relative to the potency of a reference or index chemical.
Threshold:	The dose of a chemical just sufficient to produce an effect.
TPHCWG:	Total Petroleum Hydrocarbon Criteria Working Group
TRPH:	Total Recoverable Petroleum Hydrocarbon. A means of expressing the total concentration of petroleum-related hydrocarbons in soil or water.
USEPA:	United States Environmental Protection Agency
WHO:	World Health Organization

X. Appendix A. Derivation of Body Weight, Dermal Surface Area, and Inhalation Rate Estimates

A. Introduction

As described in the technical background document for Chapter 62-777, F.A.C. (Saranko et al., 1999), body weight, surface area, and inhalation rate assumptions were previously derived from a combination of USEPA-recommended defaults and data presented in the USEPA *Exposure Factors Handbook* (USEPA, 1997). Body weight assumptions were based on standard USEPA default weights of 70 kg for an adult and 15 kg for a child. Inhalation rates for adult workers and children were also taken directly from USEPA recommendations, as was the dermal surface area assumption for workers. Data from the *Exposure Factors Handbook* were used to derive the dermal surface area assumption for the child, and also the weighted average dermal surface area and inhalation rates for the aggregate resident.

The approach in developing body weight, surface area, and inhalation rate assumptions has changed with this update. With the exception of inhalation rate in workers, standard USEPA defaults have been replaced with values derived directly from health statistics. Also, the 1997 *Exposure Factors Handbook*, which relies on data from the Second National Health and Nutrition Survey (NHANES II), is not used as the primary source of information for body weight and surface area. Instead, data from the newer NHANES III are analyzed to develop assumptions for these parameters. This change is warranted because the more recent NHANES III survey indicates that body weights have changed nationally since the NHANES II survey in the mid-1980s. Increases in body weights means that surface areas have changed as well. Use of the more recent data provides a more accurate and contemporary view of these body parameters that affect risk.

Another refinement is the manner in which body weight, surface area, and inhalation rates are developed. All three of these parameters change dramatically as an individual matures from age 1 to

age 31 years, and time averaging of each is required to derive an accurate exposure estimate, particularly for carcinogens where exposure is assumed to occur for long periods. Previously, averaging for the aggregate resident was accomplished by dividing the 30-year exposure period into two intervals — one exposure interval as a child, with fixed body weight, surface area, and inhalation rate assumptions, and the second interval as an adult with a different set of assumptions for these variables. These two sets of assumptions (child and adult) are then time-weighted to derive an average.

In this update, body weight and surface area values are developed for each age, in annual increments from ages 1 to 65 years. These values are then used to develop averages for each interval of interest. This procedure includes not only the aggregate resident (ages 1 to 31 years), but also the child resident (ages 1 to 7 years) and the adult worker (ages 18 to 65 years). This method of averaging, made possible by the more comprehensive data set available directly from NHANES III, offers more precise estimates of these exposure parameters. Age-specific inhalation rates, available from the *Exposure Factors Handbook*, are also averaged in an analogous fashion to derive inhalation rate assumptions for each scenario. Although inhalation rate data are only available for children for 2- to 3-year age intervals, and a single value is presented for adults (ages 19 to 65+ years), this averaging procedure nonetheless represents an improvement over the method of inhalation rate estimation used previously.

The updated values derived for these parameters are summarized in Table A-1 below.

Table A-1.

Summary of Body Weight, Surface Area, and Inhalation Rate Assumptions

Parameter	Exposure Scenario		
	Child	Aggregate Resident	Worker
Body Weight (kg)	16.8	51.9	76.1
Surface Area (cm^2)	2960	4810	3500
Inhalation Rate (m^3/day)	8.1	12.2	20[a]

[a] Based on Chapter 62-777, F.A.C. default.

B. Description of NHANES III

The National Center for Health Statistics (NCHS) collected vital and health statistics on 33,994 non-institutionalized individuals aged two months to 90 years old, living in the United States during 1988-1994, as part of the NHANES III. To obtain reliable estimates of characteristics of Black Americans, Mexican Americans, infants and young children (1-5 years), and older persons (60+ years), individuals in these groups were sampled at a higher rate. While this approach assisted in developing statistically valid data for these limited-size groups of special interest, it created an overall data set in which responses from these groups were over-represented relative to the U.S. population as a whole.

In order to develop data suitable for SCTL development, raw data from NHANES III are adjusted to account for non-responses and stratified to reflect the composition of the entire U.S. population by age, sex, and race using a weighting factor provided by the NCHS. NHANES III data on body weights, including clothing (estimated as ranging from 0.09 to 0.28 kg), age, sex, and race, are downloaded from the NCHS using the FERRETS data extraction tools, and converted into a Statistical Analysis System (SAS) dataset. A total of 31,311 records were available from the NHANES III data set. Those records with complete information applicable to the analysis of interest were included in the data set. Missing data accounted for the loss of 1,244 records for the body weight calculations. Mean body weights are calculated for each age grouping. Age groups are defined traditionally as starting with

the birth month and including the next 11 months. For example, age group 2 includes individuals who are 24 to 35 months old at the time of the NHANES III exam.

1. Body weights

Previous studies have shown that body weights tend to follow a lognormal distribution (Brainard and Burmaster, 1992; Burmaster and Crouch, 1997). To confirm this observation with the NHANES III data, goodness-of-fit tests are performed for each age group. These tests indicated that the lognormal assumption provides a reasonable fit for these data (results not shown). Given that the body weight data are lognormally distributed implies that:

$$\ln [BW] \sim \text{Normal } (\mu, \sigma)$$

where [BW] represents body weight in kg, and the natural logarithm transformation of the body weight (ln[BW]) is approximately normally distributed with parameters μ (mean) and σ (standard deviation).

A simple method for deriving an estimate of the mean and variance for two-parameter lognormal distributions such as this, is given by:

$$\mu = \exp\left(y + \frac{s_y^2}{2}\right)$$

$$s^2 = \mu^2[\exp(s_y^2) - 1]$$

This method, although efficient, produces estimates of the population mean and variance that may be somewhat biased. However, because of the rather large sample sizes for each age group, any bias in the resulting estimates will be small. The bias introduced into the analysis using these techniques can be estimated directly from the data by the following equation (Gilbert, 1987):

$$\text{Bias} = \left(1 - \frac{s_y^2}{n}\right)^{-(n-1)/2} \exp\left(-\frac{n-1}{2n} s_y^2\right)$$

Given that the maximum variance of the log-transformed data is generally less than 0.1 and the sample sizes are generally greater than 50, then the maximum bias introduced using this procedure will be less than 0.05%. Because the mean body weights are rounded to three significant figures, the error introduced through this method is inconsequential.

Mean and standard deviations of the body weight data for males, females, and both genders combined ('composite' body weight) for ages 1 through 31 years are given in Table A-2. It should be noted that the results for the composite body weights are not simply the average of the male and female body weights for each age group. Means for the composite body weights are generated from the raw data using the specified weighting factors that account for sample demographics including expected proportions of each sex in the population. Aggregate resident (ages 1 to 31 years) body weight for combined males and females is **51.9 kg**. The child (ages 1 to 7 years) body weight for male and female children combined is **16.8 kg**.

Workers are assumed to include, with equal probability, adults aged 18 to 65 years. The assumption that all ages in this range are equally represented in a worker population may not be correct, but the error introduced by this assumption is likely to be small. Yearly body weight estimates for male, female, and both genders combined ('composite' body weight) workers are given in Table A-3. Again, means for the composite body weights are generated from the raw data using the specified weighing factors that account for sample demographics that included expected proportions of each sex in the population. The average body weight for male and female workers aged 18-65 years is **76.1 kg**.

2. Surface areas

Limited empirical data exist for surface area measurements in adults and children. In an attempt to extend the utility of the considerable body weight data available, a number of authors have described

allometric relationships between body weight and surface area (e.g., Burmaster, 1998; Dubois and Dubois, 1916). Both univariate (based on weight only) and bivariate (based on both height and weight) models have been employed. Based on our analysis of surface areas predicted from the NHANES III dataset, these models performed equally well in predicting surface areas across a wide range of body weights (data not shown). Therefore, the univariate model proposed by Burmaster (1998) was chosen to calculate total body surface area from body weights. The advantages of this model are its inherent simplicity and the ability to extend the results to produce distributional parameters without complications resulting from confounded variables. The model is given below,

$$SA = BW^{0.681} * 1020$$

where SA is the total skin surface area (cm^2) and BW is the body weight (kg). Total body surface areas for males and females by age are listed in Table A-4.

Exposed surface area is based, in part, on guidance specified in RAGS-Part E (USEPA, 2000). Specifically, estimates of exposed surface area depend upon assumptions about the types of clothing a particular receptor population is likely to wear, and are computed by summing the area of the body parts not covered by the clothes. The percentage that each body part contributes to the total surface area is required to calculate the sum of exposed body surface area for each exposure scenario. Data on body part percentages of total surface area derived from empirical measurements of children and adults, as presented in the *Exposure Factors Handbook* (USEPA, 1989a), are used for these calculations. The number of individuals sampled to derive these data was extremely limited; sometimes as few as a single individual constitutes the sample size for an entire age group. However, no alternative source with better data was identified for this report. The percentage of total body surface area, by part, for children and adults is shown in Table A-4. No specific age group data are presented in the *Exposure Factors Handbook* for children at ages 1, 5, 7, 8, 10, 11, 14, and 15 years. Therefore, the surface area information for these ages is linearly interpolated from the adjacent age groups. Based on the

relationships in RAGS-Part E (USEPA, 2000), surface area percentage for the forearms and lower legs are assumed to equal 0.45 and 0.4 of the arm and leg, respectively.

Child surface area exposed is calculated based on a child wearing short pants, a short-sleeved shirt, and no shoes. The exposed area considered is, therefore, the head, hands, feet, lower legs and forearms. The surface area represented by each body part is calculated by multiplying the composite male/female total surface area for each age group by the percentage surface area for each body part.

$$SA_{bodypart} = (\text{Percentage Body Part for Age}) * (\text{Total Surface Area for Age})$$

The surface areas for each of the exposed body parts (head, hands, feet, lower legs, and forearms) are summed to derive a total exposed surface area for each age, as shown in Table A-6. Total surface area exposed values for each age are then averaged over the age range of interest, e.g., for a child resident, from ages 1 to 7 years. Based on this approach, the exposed surface area for a child resident is 2960 cm^2.

Aggregate resident surface area exposed is calculated in a manner similar to that for a child resident, with the exception that shoes are assumed to be worn from ages 7 to 31 years. Therefore, the exposed area considered is the head, hands, feet, lower legs and forearms for the first six years, and the head, hands, lower legs and forearms for the remaining 24 years. As above, the skin surface area for each exposed body part is calculated by multiplying its percentage relative of total body surface area by the male/female total surface area. This calculation is performed for each age group, and age-specific exposed surface areas for ages 1 to 31 years are averaged to derive the exposed surface area for the aggregate resident of **4810 cm^2**.

Worker surface area exposed is calculated based on a worker wearing long pants, shoes and a short-sleeved shirt. Therefore, the exposed area considered is the head, hands, and forearms. Surface areas for each of these exposed parts of the body, as well as the total exposed surface area, are

calculated for each age in a manner identical to the procedures described above (see Table A-7). Age-specific exposed surface areas for the workers are averaged for ages 18 to 65 to derive an exposed surface area for workers of **3500 cm^2**.

3. Inhalation rates

Inhalation rates for children and aggregate residents are based on the average daily inhalation required to support metabolism as presented in the *Exposure Factors Handbook* (Table 5-23 USEPA, 1997). Inhalation rates are given in Table A-8 for each age group. Averaging the inhalation rate for the ages 1 to 31 years produced a mean aggregate resident inhalation rate of **12.2 m^3/day**. Averaging the inhalation rates for ages 1 to 7 years produced a mean child inhalation rate of **8.1 m^3/day**. A worker inhalation rate value of **20 m^3/day** is taken from Chapter 62-777, F.A.C. (Saranko et al., 1999).

Table A-2.

Mean Body Weight Estimates for Males and Females Ages 1 to 31 Years Old.

Age	Mean Body Weights (kg)		
	Males	Females	Composite
1-2	11.6	10.9	11.2
2-3	13.6	13.2	13.4
3-4	15.8	15.4	15.6
4-5	17.6	17.8	17.7
5-6	20.1	20.1	20.1
6-7	23.2	22.5	22.9
7-8	26.3	26.4	26.3
8-9	30.1	29.8	30.0
9-10	34.4	34.3	34.3
10-11	37.3	37.9	37.6
11-12	42.4	44.1	43.3
12-13	49.1	49.0	49.0
13-14	54.0	55.8	54.8
14-15	63.8	58.4	61.1
15-16	66.8	58.2	62.0
16-17	68.6	61.6	65.3
17-18	72.8	62.3	67.8
18-19	71.2	61.4	66.2
19-20	73.0	63.7	68.2
20-21	72.5	61.7	66.2
21-22	72.9	64.9	69.0
22-23	76.6	64.0	69.8
23-24	77.8	66.8	72.6
24-25	78.5	62.7	70.6

Table A-2. Continued

Age	Mean Body Weights (kg)		
	Males	Females	Composite
25-26	80.2	66.2	74.4
26-27	75.8	64.7	69.6
27-28	81.2	65.0	73.6
28-29	80.8	67.0	73.7
29-30	81.8	66.0	74.0
30-31	83.4	67.6	75.2
Average Aggregate Resident (1 to 31 years) Body Weight			**51.9**
Average Child (1 to 7 years) Body Weight			**16.8**

Table A-3.

Mean Body Weight Estimates for Males and Females Ages 18 to 65 Years Old.

Age	Mean Male Body Weight (kg)	Mean Female Body Weight (kg)	Composite Body Weight (kg)
18-19	71.2	61.4	66.2
19-20	73.0	63.7	68.2
20-21	72.5	61.7	66.2
21-22	72.9	64.9	69.0
22-23	76.6	64.0	69.8
23-24	77.8	66.8	72.6
24-25	78.5	62.7	70.6
25-26	80.2	66.2	74.4
26-27	75.8	64.7	69.6
27-28	81.2	65.0	73.6
28-29	80.8	67.0	73.7
29-30	81.8	66.0	74.0
30-31	83.4	67.6	75.2
31-32	79.5	72.6	76.4
32-33	81.6	67.5	74.3
33-34	83.9	68.3	75.2
34-35	83.1	67.4	76.8
35-36	81.5	71.4	76.0
36-37	87.5	65.9	78.3
37-38	83.2	72.0	76.4
38-39	82.4	71.6	76.6
39-40	82.6	74.6	78.7
40-41	85.8	68.5	75.7

Table A-3. Continued

Age	Mean Male Body Weight (kg)	Mean Female Body Weight (kg)	Composite Body Weight (kg)
41-42	86.3	70.0	79.0
42-43	85.1	72.6	78.9
43-44	86.4	68.8	78.1
44-45	90.6	72.5	79.4
45-46	83.6	71.7	78.0
46-47	80.8	72.0	76.2
47-48	85.5	72.0	79.4
48-49	82.3	75.8	79.0
49-50	82.1	73.3	77.6
50-51	81.7	73.8	76.9
51-52	85.6	79.5	83.1
52-53	87.1	72.0	79.8
53-54	89.3	73.8	81.7
54-55	86.0	74.5	79.6
55-56	83.0	72.6	76.7
56-57	87.1	77.6	82.9
57-58	86.3	75.6	81.7
58-59	83.4	72.2	76.8
59-60	87.9	73.9	80.5
60-61	83.5	68.9	76.0
61-62	81.8	72.1	76.2
62-63	82.0	72.8	76.7
63-64	84.4	71.3	76.9
64-65	84.3	74.5	78.7
Average Worker (18 to 65 years) Body Weight			**76.1**

Table A-4.

Surface Area for Males and Females Based on Body Weight Estimates

Age	Total Surface Area (cm^2)		
	Male	Female	Composite
1-2	5390	5170	5280
2-3	6020	5890	5960
3-4	6660	6550	6610
4-5	7190	7230	7210
5-6	7840	7860	7850
6-7	8640	8470	8560
7-8	9410	9410	9410
8-9	10320	10240	10290
9-10	11280	11240	11260
10-11	11930	12040	11980
11-12	13010	13370	13190
12-13	14380	14350	14360
13-14	15330	15680	15500
14-15	17150	16200	16690
15-16	17750	16180	16880
16-17	18060	16790	17470
17-18	18850	16940	17940
18-19	18550	16740	17630
19-20	18880	17170	17990
20-21	18790	16810	17640
21-22	18880	17380	18130
22-23	19490	17250	18280
23-24	19720	17740	18770

Table A-4. Continued

Age	Total Surface Area (cm^2)		
	Male	Female	Composite
24-25	19820	17010	18420
25-26	20100	17610	19060
26-27	19380	17360	18240
27-28	20300	17410	18940
28-29	20190	17780	18940
29-30	20380	17610	19000
30-31	20660	17870	19200
31-32	20010	18740	19440
32-33	20360	17840	19060
33-34	20750	18000	19210
34-35	20610	17870	19510
35-36	20330	18540	19350
36-37	21310	17590	19720
37-38	20620	18650	19420
38-39	20500	18570	19460
39-40	20560	19100	19830
40-41	21080	18050	19300
41-42	21120	18330	19870
42-43	20940	18730	19850
43-44	21160	18110	19720
44-45	21830	18740	19930
45-46	20720	18620	19730
46-47	20250	18680	19420
47-48	21010	18680	19950
48-49	20490	19340	19920

Table A-4. Continued

Age	Total Surface Area (cm^2)		
	Male	Female	Composite
49-50	20450	18870	19640
50-51	20390	18980	19520
51-52	21040	19960	20590
52-53	21310	18660	20030
53-54	21680	18980	20340
54-55	21100	19070	19960
55-56	20610	18810	19520
56-57	21310	19650	20570
57-58	21160	19280	20350
58-59	20670	18700	19510
59-60	21420	19020	20150
60-61	20700	18140	19380
61-62	20400	18700	19410
62-63	20430	18800	19490
63-64	20850	18560	19530
64-65	20820	19100	19830

Table A-5.

Percentage Surface Area by Body Part*

Age	Surface Area (%)						
	Head	**Arms**	**Hands**	**Legs**	**Feet**	***Forearms***	***Lower legs***
0-1	**18.20**	**13.70**	**5.30**	**20.60**	**6.54**	*6.17*	*8.24*
1-2	**16.50**	**13.00**	**5.68**	**23.10**	**6.27**	*5.85*	*9.24*
2-3	**14.20**	**11.80**	**5.30**	**23.20**	**7.07**	*5.31*	*9.28*
3-4	**13.60**	**14.40**	**6.07**	**26.80**	**7.21**	*6.48*	*10.72*
4-5	**13.80**	**14.00**	**5.70**	**27.80**	**7.29**	*6.30*	*11.12*
5-6	*13.45*	*13.55*	*5.21*	*27.45*	*7.10*	*6.10*	*10.98*
6-7	**13.10**	**13.10**	**4.71**	**27.10**	**6.90**	*5.90*	*10.84*
7-8	*12.73*	*12.83*	*4.91*	*27.63*	*7.13*	*5.78*	*11.05*
8-9	*12.37*	*12.57*	*5.10*	*28.17*	*7.35*	*5.66*	*11.27*
9-10	**12.00**	**12.30**	**5.30**	**28.70**	**7.58**	*5.54*	*11.48*
10-11	*10.91*	*12.77*	*5.33*	*29.30*	*7.40*	*5.75*	*11.72*
11-12	*9.83*	*13.23*	*5.36*	*29.90*	*7.21*	*5.96*	*11.96*
12-13	**8.74**	**13.70**	**5.39**	**30.50**	**7.03**	*6.17*	*12.20*
13-14	**9.97**	**12.10**	**5.11**	**32.00**	**8.02**	*5.45*	*12.80*
14-15	*9.30*	*12.43*	*5.30*	*32.53*	*7.66*	*5.60*	*13.01*
15-16	*8.63*	*12.77*	*5.49*	*33.07*	*7.29*	*5.75*	*13.23*
16-17	**7.96**	**13.10**	**5.68**	**33.60**	**6.93**	*5.90*	*13.44*
17-18	**7.58**	**17.50**	**5.13**	**30.80**	**7.28**	*7.88*	*12.32*
18-65	**6.64**	**14.35**	**4.98**	**32.67**	**6.75**	*6.46*	*13.07*

* Values in **bold** are taken directly from the EFH, values in *italics* are derived as specified in the text.

Table A-6.

Exposed Surface Areas for Child and Aggregate Residents

Age	Body Part Surface Area (cm^2)					Surface Area (cm^2)
	Head	Hands	Feet	Forearms	Lower Legs	Total Exposed
1-2	871.2	299.9	331.1	308.9	487.9	2299
2-3	846.3	315.9	421.4	316.5	553.1	2453
3-4	899.0	401.2	476.6	428.3	708.6	2914
4-5	995.0	411.0	525.6	454.2	801.8	3188
5-6	1055.8	408.6	557.0	478.7	861.9	3362
6-7	1121.4	403.2	590.6	504.6	927.9	3548
7-8	1198.2	461.7		543.4	1040.1	3244
8-9	1272.5	525.1		581.9	1159.3	3539
9-10	1351.2	596.8		623.2	1292.6	3864
10-11	1307.4	638.5		688.3	1404.1	4038
11-12	1296.1	707.0		785.5	1577.5	4366
12-13	1255.1	774.0		885.3	1751.9	4666
13-14	1545.4	792.1		844.0	1984.0	5165
14-15	1552.2	884.6		933.8	2171.9	5543
15-16	1456.7	926.7		969.8	2232.7	5586
16-17	1390.6	992.3		1029.9	2348.0	5761
17-18	1359.9	920.3		1412.8	2210.2	5903
18-19	1170.6	878.0		1138.5	2303.9	5491
19-20	1194.5	895.9		1161.7	2350.9	5603
20-21	1171.3	878.5		1139.1	2305.2	5494
21-22	1203.8	902.9		1170.7	2369.2	5647
22-23	1213.8	910.3		1180.4	2388.8	5693
23-24	1246.3	934.7		1212.1	2452.9	5846

Table A-6. Continued

Age	Body Part Surface Area (cm^2)					Surface Area (cm^2)
	Head	Hands	Feet	Forearms	Lower Legs	Total Exposed
24-25	1223.1	917.3		1189.5	2407.1	5737
25-26	1265.6	949.2		1230.8	2490.8	5936
26-27	1211.1	908.4		1177.8	2383.6	5681
27-28	1257.6	943.2		1223.1	2475.1	5899
28-29	1257.6	943.2		1223.1	2475.1	5899
29-30	1261.6	946.2		1226.9	2482.9	5917
30-31	1274.9	956.2		1239.8	2509.1	5980
Average Child (1 to 7 years) Surface Area						**2960***
Average Aggregate Resident (1 to 31 years) Surface Area						**4810***

* Final surface area rounded to three significant figures.

Table A-7.

Exposed Surface Areas for Workers

Age	Surface Area for Body Part (cm^2)			Surface Area (cm^2)
	Head	Hands	Forearms	Total Exposed
18-19	1170.6	878.0	1138.5	3187
19-20	1194.5	895.9	1161.7	3252
20-21	1171.3	878.5	1139.1	3189
21-22	1203.8	902.9	1170.7	3277
22-23	1213.8	910.3	1180.4	3305
23-24	1246.3	934.7	1212.1	3393
24-25	1223.1	917.3	1189.5	3330
25-26	1265.6	949.2	1230.8	3446
26-27	1211.1	908.4	1177.8	3297
27-28	1257.6	943.2	1223.1	3424
28-29	1257.6	943.2	1223.1	3424
29-30	1261.6	946.2	1226.9	3435
30-31	1274.9	956.2	1239.8	3470
31-32	1290.8	968.1	1255.3	3514
32-33	1265.6	949.2	1230.8	3446
33-34	1275.5	956.7	1240.5	3473
34-35	1295.5	971.6	1259.9	3527
35-36	1284.8	963.6	1249.5	3498
36-37	1309.4	982.1	1273.4	3565
37-38	1289.5	967.1	1254.0	3511
38-39	1292.1	969.1	1256.6	3518
39-40	1316.7	987.5	1280.5	3585
40-41	1281.5	961.1	1246.3	3489

Table A-7. Continued

Age	Surface Area for Body Part (cm^2)			Surface Area (cm^2)
	Head	Hands	Forearms	Total Exposed
41-42	1319.4	989.5	1283.1	3592
42-43	1318.0	988.5	1281.8	3588
43-44	1309.4	982.1	1273.4	3565
44-45	1323.4	992.5	1287.0	3603
45-46	1310.1	982.6	1274.1	3567
46-47	1289.5	967.1	1254.0	3511
47-48	1324.7	993.5	1288.3	3606
48-49	1322.7	992.0	1286.3	3601
49-50	1304.1	978.1	1268.3	3550
50-51	1296.1	972.1	1260.5	3529
51-52	1367.2	1025.4	1329.6	3722
52-53	1330.0	997.5	1293.4	3621
53-54	1350.6	1012.9	1313.5	3677
54-55	1325.3	994.0	1288.9	3608
55-56	1296.1	972.1	1260.5	3529
56-57	1365.8	1024.4	1328.3	3719
57-58	1351.2	1013.4	1314.1	3679
58-59	1295.5	971.6	1259.9	3527
59-60	1338.0	1003.5	1301.2	3643
60-61	1286.8	965.1	1251.5	3503
61-62	1288.8	966.6	1253.4	3509
62-63	1294.1	970.6	1258.6	3523
63-64	1296.8	972.6	1261.1	3531
64-65	1316.7	987.5	1280.5	3585
Average Worker (18 to 65 years) Surface Area				**3500***

* Final surface area rounded to three significant figures.

Table A-8.

Inhalation Rates for Child and Adult Residents Ages 1 to 31 Years.

Age	Inhalation Rate (m^3/day)		
	Male	Female	Average Male and Female
1-2	6.8	6.8	6.8
2-3	6.8	6.8	6.8
3-4	8.3	8.3	8.3
4-5	8.3	8.3	8.3
5-6	8.3	8.3	8.3
6-7	10	10	10
7-8	10	10	10
8-9	10	10	10
9-10	14	13	13.5
10-11	14	13	13.5
11-12	14	13	13.5
12-13	15	12	13.5
13-14	15	12	13.5
14-15	15	12	13.5
15-16	17	12	14.5
16-17	17	12	14.5
17-18	17	12	14.5
18-19	17	12	14.5
19-20	15.2	11.3	13.25
20-21	15.2	11.3	13.25
21-22	15.2	11.3	13.25
22-23	15.2	11.3	13.25

Table A-8. Continued

Age	Inhalation Rate (m^3/day)		
	Male	Female	Average Male and Female
23-24	15.2	11.3	13.25
24-25	15.2	11.3	13.25
25-26	15.2	11.3	13.25
26-27	15.2	11.3	13.25
27-28	15.2	11.3	13.25
28-29	15.2	11.3	13.25
29-30	15.2	11.3	13.25
30-31	15.2	11.3	13.25
Aggregate Resident (1 to 31 years) Inhalation Rate			**12.2***
Child Resident (1 to 7 years) Inhalation Rate			**8.1***

* Final inhalation rate rounded to one decimal place.

XI. Appendix B: Derivation of Inhalation and Dermal Toxicity Values

A. Inhalation Toxicity Values

For evaluating hazard from the inhalation of a chemical of concern, the USEPA develops toxicity values in the form of Reference Doses (RfDs) or Reference Concentrations (RfCs). While the USEPA has recently shown preference for RfCs, the equations employed and methods described in this report use RfDs exclusively. The reason for using RfDs instead of RfCs is that children have much higher ventilation rates relative to body weight than adults. Consequently, children exposed to air with a given RfC will receive a higher dosage from air than an adult. While the use of RfDs allows this difference to be taken into consideration, the use of RfCs involves the implicit assumption that adults and children are equally sensitive to contamination in air. The same rationale explains the use in the equation for carcinogenicity of Inhalation Slope Factors (ISFs) rather than Inhalation Unit Risk (IUR) values (which are expressed as recognized air concentrations).

In situations where the USEPA lists both an inhalation RfD and an inhalation RfC for a noncarcinogen or, alternatively, an ISF and an IUR for a carcinogen, the listed RfD or ISF in question has been converted from the RfC or IUR, respectively. The USEPA reports these converted toxicity values to one significant figure for inhalation RfDs or two significant figures for ISFs. In the development of the CTLs, inhalation RfDs and ISFs converted from RfCs and IUR without rounding of the final value are used in preference to the rounded USEPA inhalation RfDs or ISFs.

1. Reference Dose (RfD)

When an inhalation RfC is available, it is converted to an inhalation RfD for the calculation of a soil CTL. The conversion from RfC to inhalation RfD assumed a 70 kg individual breathing 20 m^3/day. Thus, the RfC is multiplied by 20 m^3/day and divided by 70 kg to obtain a value with the units mg/kg/day. The final value is not rounded.

e.g., Methyl *tert*-butyl ether: Inhalation RfC = 3 mg/m^3

thus, RfD_i = (3 mg/m^3 x 20 m^3/day) / 70 kg = 8.57142857 x 10^{-1} mg/kg/day

When an RfC is not available, the second choice is to develop an inhalation RfD from the oral RfD using route-to-route extrapolation. Such extrapolation is only done when the toxic endpoint being addressed is systemic in nature. Oral RfDs that are known or likely to be route-specific (e.g., where the toxic endpoint involved the gastrointestinal tract) are not extrapolated.

The formula for the conversion of an oral RfD to an inhalation RfD is as follows:

$$RfD_i = RfD_o \times GI\ Absorption$$

e.g., Anthracene: RfD_o = 3.0 x 10^{-1} mg/kg/day

Chemical Specific GI Abs Factor = 0.5

thus, (3.0 x 10^{-1} mg/kg/day) x (0.5) = 1.5 x 10^{-1} mg/kg/day

2. Slope Factor (SF)

When a carcinogen had an inhalation unit risk (IUR), the IUR is converted to an ISF for the calculation of a soil target level. The conversion assumes a 70 kg individual breathing 20 m^3/day. Thus, the IUR (Unit Risk/$\mu g/m^3$) is divided by 20 m^3/day and multiplied by 70 kg and a conversion factor of 1000 µg/mg to obtain a value with the units $(mg/kg/day)^{-1}$. The final value is not rounded.

e.g., Benzene: IUR = 7.8 x 10^{-6} UR/$\mu g/m^3$

thus, ISF = [((7.8 x 10^{-6} UR/$\mu g/m^3$) / 20m^3/day) x 70 kg x 1000 µg/mg] =

= 2.73 x 10^{-2} $(mg/kg/day)^{-1}$

If an IUR is not available and the chemical is regarded as likely producing carcinogenicity via a systemic effect, an ISF is derived from the oral slope factor (OSF), if available. This route-to-route extrapolation is accomplished by using the following formula:

$$ISF = OSF / GI\ Absorption$$

In general, route-to-route extrapolation from the OSF is not performed if the OSF is known or presumed to reflect route-specific toxicity. When a chemical exhibits route-specific toxicity, it exerts its toxic effect (i.e., cancer) only by a specific exposure route. For example, chromium only causes lung cancer if it is inhaled, thus the toxic effect (lung cancer) is route-specific and target organ-specific. No other exposure route for chromium has been shown to cause cancer.

B. Dermal Toxicity Values

1. Reference Dose (RfD)

Dermal RfDs are derived from either the oral or inhalation RfD (if both are available and suitable, preference is given to the oral RfD). The following formula is used:

$$RfD_d = RfD_o \times GI\ Absorption$$

If an RfD (either oral or inhalation) is known or presumed to be route-specific, it is not regarded as suitable for route-to-route extrapolation.

2. Cancer Slope Factor (SF)

Dermal cancer slope factors (DSFs) are derived from OSFs using route-to-route extrapolation:

$$DSF = OSF / GI\ Absorption$$

e.g., Benzene: $OSF = 2.9 \times 10^{-2}$ $(mg/kg/day)^{-1}$

Chemical-Specific GI Abs = 0.9

thus, $DSF = (2.9 \times 10^{-2}\ (mg/kg/day)^{-1}) \div (0.9) = 3.2 \times 10^{-2}\ (mg/kg/day)^{-1}$

In general, OSFs are not extrapolated to produce DSFs if they are thought to reflect route-specific toxicity. In the case of carcinogenic PAHs the toxic endpoint (cancer) occurs regardless of the route of exposure. This effect is clearly evidenced by the fact that while the OSF for benzo(a)pyrene is based on data in which oral dosing resulted in GI tract tumors in rodents, arguably a route-specific cancer, benzo(a)pyrene has also been observed to produce other types of cancer in several species when administered by a variety of routes, including inhalation and dermal contact. Although no cancer slope factor has yet been derived for these routes, the rather strong evidence that benzo(a)pyrene (and, by implication, other carcinogenic PAHs) is carcinogenic by a variety of routes, indicates that PAH-induced cancer is not wholly route-specific. Because of this property, route-to-route extrapolation is performed to derive both inhalation and dermal slope factors from the OSF for this group of chemicals.

XII. Appendix C: Technical Basis for TRPH CTLs

The following calculations for total recoverable petroleum hydrocarbon (TRPH) values are adopted essentially as described in the Total Petroleum Hydrocarbon Criteria Working Group (TPHCWG, 1997a,b,c; Volumes III and IV, and the Technical Overview).

The application of a general standard for TRPHs is difficult because of the variation in mobility and toxicity of the chemicals included. To overcome this problem, TPHCWG (1997a) suggests a sub-classification methodology in which aromatics and aliphatics are considered separately because these groups vary considerably in their environmental behavior. Each of these groups is then further subdivided on the basis of equivalent carbon number index (EC). The EC is a function of the molecular weight (MW) and boiling point (BP) of a chemical normalized to the BP of the n-alkanes, or its retention time in a BP gas chromatographic column. This approach is used since it is consistent with methods routinely used in the petroleum industry for separating complex mixtures and is a more appropriate differentiation technique than the actual carbon number of the chemical.

Table C-1.

Hydrocarbon Fractions Defined by the Total Petroleum Hydrocarbon Criteria Working Group

Range of Equivalent Carbon Number (EC)	Avg EC	Classification
C_5-C_7	6.5	Aromatic
>C_7-C_8	7.5	Aromatic
>C_8-C_{10}	9.0	Aromatic
>C_{10}-C_{12}	11	Aromatic
>C_{12}-C_{16}	14	Aromatic
>C_{16}-C_{21}	18.5	Aromatic
>C_{21}-C_{35}	28.5	Aromatic
C_5-C_6	5.5	Aliphatic
>C_6-C_8	7.0	Aliphatic
>C_8-C_{10}	9.0	Aliphatic
>C_{10}- C_{12}	11	Aliphatic
>C_{12}- C_{16}	14	Aliphatic
>C_{16}- C_{21}	18.5	Aliphatic

A. Calculation of TRPH Fraction-Specific Physical Properties

Several alternatives for estimating representative physical/chemical properties for each fraction were reviewed by the TPHCWG. They included simple averaging of all available property data, composition-based averaging in which a weighted average of the available property data was computed based on the relative mass of each component in gasoline, and correlation to relative boiling point index in which the properties were developed based on EC values. While all of the approaches had similar results, it is determined that the correlations approach is most useful, because if the definition of the fractions change, new properties can be easily computed.

Utilizing the values correlations approach, the TRPHs are grouped into EC fractions, a method which allows for the calculation of the fate and transport characteristics of solubility (S), organic carbon partition coefficient (K_{oc}) and vapor pressure (atm). While Henry's Law constant (HLC) could also be estimated from a similar type of equation, TPHCWG determined that using the estimated molecular weights, solubilities and vapor pressures to calculate HLC allowed for internal consistency with the other estimated values. The formulas provided by TPHCWG (1997a) are as follows:

Aromatics:

$\text{Log } S = (-0.21 \times EC) + 3.7$

$\text{Log } K_{oc} = (0.10 \times EC) + 2.3$

Aliphatics:

$\text{Log } S = (-0.55 \times EC) + 4.58$

$\text{Log } K_{oc} = (0.45 \times EC) + 0.43$

Aliphatics and Aromatics

$\text{Log VP} = (-0.5 \times EC) + 2.3 \quad [\text{for EC} \leq 12]$

$\text{Log VP} = (-0.36 \times EC) + 0.72 \quad [\text{for EC} > 12]$

$$\text{H' (unitless)} = \frac{\text{Vapor Pressure (atm)} \times \text{Molecular Weight (g/mol)}}{\text{Solubility (mg/L)} \times 8.2\text{x}10^{-5}\ (\text{atm - m}^3\text{/mol - K}) \times 293\text{K}}$$

Henry's Law constant (atm-m3/mol) = H' (unitless)/41

When diffusivity in air or water was plotted as a function of equivalent carbon number, TPHCWG found that the values did not vary significantly from compound to compound. Thus, a conservative, reasonable assumption is to set $D_{air} = 10^{-1}$ cm^2/sec and $D_{water} = 10^{-5}$ cm^2/sec for all fractions.

Using the models above, the following chemical values for the TRPH classes have been assigned:

Table C-2.

Assigned Chemical Properties of TRPH Classes Based on an Equivalent Carbon Number[a]

TRPH Class	Avg. EC	Proposed Value				
		H(atm-m^3/mol)[a]	MW (g/mol)	K_{oc} (mL/g)[b]	S (mg/L)[b]	VP (atm)[b]
C_5-C_7 Aromatic	6.5	5.61 E-3	NC[c]	NC	NC	NC
>C_7-C_8 Aromatic	7.5	6.64 E-3	NC	NC	NC	NC
>C_8-C_{10} Aromatic	9.0	1.17 E-2	1.2 E+2	1.58 E+3	6.5 E+1	6.3 E-3
>C_{10}-C_{12} Aromatic	11	3.41 E-3	1.3 E+2	2.51 E+3	2.5 E+1	6.3 E-4
>C_{12}-C_{16} Aromatic	14	1.29 E-3	1.5 E+2	5.01 E+3	5.8 E+0	4.8 E-5
>C_{16} -C_{21} Aromatic	18.5	3.17 E-4	1.9 E+2	1.58 E+4	6.5 E-1	1.1 E-6
>C_{21} -C_{35} Aromatic	28.5	1.63 E-5	2.4 E+2	1.26 E+5	6.6 E-3	4.4 E-10
C_5-C_6 Aliphatic	5.5	8.05 E-1	8.1 E+1	7.94 E+2	3.6 E+1	3.5 E-1
>C_6-C_8 Aliphatic	7.0	1.22 E+0	1.0 E+2	3.98 E+3	5.4 E+0	6.3 E-2
>C_8-C_{10} Aliphatic	9.0	1.93 E+0	1.3 E+2	3.16 E+4	4.3 E-1	6.3 E-3
>C_{10}-C_{12} Aliphatic	11	2.93 E+0	1.6 E+2	2.51 E+5	3.4 E-2	6.3 E-4
>C_{12}-C_{16} Aliphatic	14	1.29 E+1	2.0 E+2	5.01 E+6	7.6 E-4	4.8 E-5
>C_{16}-C_{21} Aliphatic	18.5	1.20 E+2	2.7 E+2	6.30 E+8	2.5 E-6	1.1 E-6

[a] Calculated using methods described above. H' (unitless) is calculated according to the formula presented above. Final values rounded to two significant figures.

[b] Calculated according to formulas in Tables 7, 9, and 12 of TPHCWG 1997a.

[c] Values for the C_5-C_7 and >C_7-C_8 aromatics, which correspond to benzene and toluene, are not calculated according to the TPHCWG methods. Chemical-specific values for these fractions are assumed to be equal to those of benzene and toluene, thus the K_{oc} and H values from Table 3a of the Technical Report are used.

Table C-3.

Calculated Chemical Properties of TRPH Classes

TRPH Class	Calculated Fraction-Specific Values[a]		
	D_a(cm^2/sec)	Volatilization Factor[b] (m^3/kg)	
		Residential	Industrial
C_5-C_7 Aromatic	2.16753 E-3	3.34080 E+3	3.04971 E+3
>C_7-C_8 Aromatic	1.01478 E-3	4.88255 E+3	4.45713 E+3
>C_8-C_{10} Aromatic	2.64276 E-4	9.56760 E+3	8.73399 E+3
>C_{10}-C_{12} Aromatic	4.90522 E-5	2.22077 E+4	2.02727 E+4
>C_{12}-C_{16} Aromatic	9.34192 E-6	5.08878 E+4	4.64540 E+4
>C_{16} -C_{21} Aromatic	7.30304 E-7	1.82004 E+5	1.66146 E+5
>C_{21} -C_{35} Aromatic	4.79300 E-9	2.24661 E+6	2.05087 E+6
C_5-C_6 Aliphatic	1.58243 E-2	1.23643 E+3	1.12870 E+3
>C_6-C_8 Aliphatic	7.96707 E-3	1.74254 E+3	1.59071 E+3
>C_8-C_{10} Aliphatic	2.05971 E-3	3.42712 E+3	3.12852 E+3
>C_{10}-C_{12} Aliphatic	4.18629 E-4	7.60182 E+3	6.93948 E+3
>C_{12}-C_{16} Aliphatic	9.34285 E-5	1.60913 E+4	1.46893 E+4
>C_{16}-C_{21} Aliphatic	6.93277 E-6	5.90716 E+4	5.39247 E+4

[a] All calculations carried out to 18 decimal places. Values provided have been rounded for presentation in this table.

[b] For residential exposure to non-carcinogens, VFs are based on an exposure duration of six years. Industrial exposure duration is 25 years.

B. Derivation of TRPH Fraction Toxicological Values

The toxicity values for the various TRPH fractions were obtained from TPHCWG (1997b) and are as follows:

Table C-4.

Toxicity Values of TRPH Classes[a]

TRPH Class	GI Absorption (%)[b]	RfD_o (mg/kg-day)	RfD_d (mg/kg-day)[c]	RfD_i (mg/kg-day)[d]
C5-C7 Aromatic	90	0.2	0.18	0.1143
>C7-C8 Aromatic	80	0.2	0.16	0.1143
>C8-C10 Aromatic	50	0.04	0.020	0.05714
>C10-C12 Aromatic	50	0.04	0.020	0.05714
>C12-C16 Aromatic	50	0.04	0.020	0.05714
>C16 -C21 Aromatic	50	0.03	0.015	0.015[e]
>C21 -C35 Aromatic	50	0.03	0.015	0.015[e]
C5-C6 Aliphatic	50	5.0	2.5	5.257
>C6-C8 Aliphatic	50	5.0	2.5	5.257
>C8-C10 Aliphatic	50	0.1	0.05	0.2857
>C10-C12 Aliphatic	50	0.1	0.05	0.2857
>C12-C16 Aliphatic	50	0.1	0.05	0.2857
>C16-C35 Aliphatic	50	2.0	1.0	1.0[e]

[a] Toxicity Values from TPHCWG (1997c).
[b] Based on ATSDR Toxicological Profile for TPH.
[c] RfD_d values extrapolated from RfD_o, using fraction-specific GI absorption (see Appendix B).
[d] RfD_i values extrapolated from RfC_i values when available (see Appendix B).
[e] RfD_i values extrapolated from RfD_o, using fraction-specific GI absorption (see Appendix B).

C. Derivation of TRPH CTLs

The DERM TRPH CTLs will be based on a 2-tiered approach. First, there will be a primary TRPH CTL. This CTL is based on the assumption that the TRPHs consist exclusively of aromatic hydrocarbons in the >C8-C10 range. Second, if the primary CTL is exceeded, then the TRPHs may be subclassified with each class possessing its own CTL. Given the potential for the subclassification methodology to yield relatively high CTLs, it is possible that the human health CTLs for some

constituents, particularly those with relatively low toxicity and low mobility potential (such as TRPHs) could result in staining, odor and/or nuisance conditions.

The primary TRPH CTL is based on the >C8-C10 carbon range as a result of two factors. First, the analytical method identified by DERM for the purpose of measuring petroleum hydrocarbons in water and soil is limited to the detection of products within a carbon chain range of C8-C40. This method, the Florida Petroleum Residual Organic (FL-PRO) — Alternative Method to Total Petroleum Hydrocarbons, 418.1 or 9073 — combines several of the commonly used methods so that the targeted range of petroleum hydrocarbons can be analyzed in a single step. However, because of its limitations, the smallest detectable C-range using the FL-PRO method is the >C8-C10 grouping. Secondly, the TRPH CTL value is selected based on the identification of the most conservative values. The calculation of the CTLs (listed below) using standard DERM and USEPA protocols results in the most conservative values for the C5-C7 aromatics. However, due to the limitations of the TRPH method of analysis, and since the most toxic and prevalent COCs within this range are addressed by other analyses and individual CTLs, the values in this group are not used as TRPH CTLs. The next most conservative values for residential and industrial direct exposure that occur within a carbon range that can be analyzed by FL-PRO are found in the >C8-C10 aromatics grouping. Therefore, the TRPH CTL values are based on this group of total petroleum hydrocarbons.

D. Calculation of the CTLs

With the assignment of the above chemical and toxicological values, the determination of risk-based CTLs follows the same methodology as that used for individual compounds.

Table C-5.

Calculated CTLs for TRPH Classes

TRPH Class	CTL (mg/kg_{soil})		
	Residential	Industrial	Leachability[a]
C_5-C_7 Aromatic	360	1900	34
>C_7-C_8 Aromatic	520	2800	59
>C_8-C_{10} Aromatic	**460**	**2700**	**340**
>C_{10}-C_{12} Aromatic	900	5900	520
>C_{12}-C_{16} Aromatic	1500	12000	1000
>C_{16} -C_{21} Aromatic	1300	11000	3200
>C_{21} -C_{35} Aromatic	2300	40000	25000
C_5-C_6 Aliphatic	6200	33000	470
>C_6-C_8 Aliphatic	8700	46000	1300
>C_8-C_{10} Aliphatic	850	4800	7000
>C_{10}-C_{12} Aliphatic	1700	10000	51000
>C_{12}-C_{16} Aliphatic	2900	21000	*
>C_{16}-C_{35} Aliphatic	42000	280000	*

[a] Based on an acceptable groundwater concentration of 5000 µg/L.
* Not a health concern for this exposure scenario.

XIII. Figures & Tables